

Matary Textbook of

GENERAL SURGERY

Mohammed El-Matary



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Dedication

Allah the all merciful, I beg Thee
To accept this effort
For the soul of my mother
She was your gift for me

Acknowledgement

The author wishes to acknowledge with gratitude:

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Preface

This book provides an update for medical students who need to keep abreast of recent developments. I hope also it will be useful for those preparing for postgraduate examination.

This book is designed to provide a concise summary of breast, endocrine surgery & general surgery, which medical students and others can use as study guide by itself or with readings in current textbooks, monographs, and reviews.

Summaries of relevant anatomical considerations are included in every chapter, taking into account that this book is written primarily for those who have some knowledge of anatomy, physiology, biochemistry and pharmacology.

The author is extremely grateful to all the contributors for the high standard of the new chapters, and hopes that you, the reader, will enjoy going through these pages as much as he had.

M. El-Matary

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Important Keys

1- **NB:** is put in a rectangular like that:



2- **Applied anatomy** is put in a rectangular like that:



3- **Oral questions** are put in rectangular like that:



4- Data which is important in **MCQs questions for Ain Shams University** is written in **blue color** like that (**bleeding....**)

5- (★) indicates the reference (look at the bottom of the page).

Abbreviations

AJCC: American Joint Committee of Cancer
ANDI: Aberration of Normal Development and Involution
A-V: Arteriovenous
ARDS: Adult Respiratory Distress Syndrome
AXR: Abdominal X-ray
BP: Blood Pressure
CA: Cancer Antigen
Ca: Calcium
CBC: Complete Blood Picture
CCA: Common Carotid Artery
CEA: Carcino-embryonic Antigen
CNS: Central Nervous System
CMF: Cyclophosphamide, Methotrexate, 5- Fluorouracil
CRP: C-reactive protein
C & S: Culture and Sensitivity
CT: Computerized Topography
CXR: Chest X-ray
DD: Differential Diagnosis
DCIS: Ductal Carcinoma In Situ
DIC: Disseminated Intravascular Coagulopathy
DM: Diabetes Mellitus
ECG: Electrocardiogram
ESR: Erythrocyte Sedimentation Rate
FNABC: Fine Needle Aspiration Biopsy and Cytology
GFR: Glomerular Filtration Rate.
HTN: Hypertension
ICA: Internal Carotid Artery
IJV: Internal Jugular Vein
IV: Intravenous
IVC: Inferior Vena Cava
IVU: Intravenous urography
KFT: Kidney Function Tests
LCIS: Lobular Carcinoma In Situ
LN: Lymph Node
MEN: Multiple Endocrine Neoplasia
MRI: Magnetic Resonance Imaging
PT: Prothrombin Time
TB: Tuberculosis
TLC: Total Leucocytic count
TRAM: Transverse Rectus Abdominis Myocutaneous Flap
TTT: Treatment
UK: United Kingdom
UO: Urine Output
U/S: Ultrasound

CHAPTER 1

THE BREAST

Anatomy of the Breast

➡ See surgical anatomy book.

Congenital Anomalies

1. Anomalies of the Nipple

⇒ **Athelia:** (very rare)

- Absence of the **nipple**.
- Usually associated with absent breast (**amazia**).

⇒ **Polythelia:**

- **Multiple nipples along either or both milk lines** (from axillae to groins)
- **Incidence: 2-6% of human females have accessory nipple.**
- **DD:** Cample de Morgan spots, mole or wart.

⇒ **Congenital retraction of the nipple:**

- It must be differentiated from acquired retraction

	Congenital Retraction	Acquired retraction
History	Since Birth	Recent
Side	Bilateral	Unilateral
Mass	No breast mass	Presence of breast mass
Pulling	Can be pulled	Can not be pulled

- **TTT:**

- Frequent pulling of nipple
- Ashford's operation: purse string suture around the nipple

⇒ **Causes of acquired nipple retraction:**

1. **Cancer breast** → circumfrential retraction.
2. **Mammary duct ectasia.**
3. **Chronic breast abscess or chronic inflammation (eg.TB).**
4. **Retraction at puberty (simple nipple inversion) of unknown etiology (bilateral in 25% of cases).**
5. **Duct ectasis** → slit like nipple retraction.



Congenital nipple retraction & accessory nipple



Nipple retraction due to cancer breast

2. Anomalies of the Breast

⇒ **Amastia** :a condition where breast tissue, nipple, and areola is absent

Poland syndrome: Athelia or amastia is sometimes associated with (i.e., absent sternal portion of pectoralis major muscle, absence of ribs 2-5, deformities of hands or vertebrae). It is more common in males.

⇒ **Amazia:**

- Absence of the mammary gland but the nipple and areola remain present (usually unilateral)
- Usually associated with absent sternal portion of pectoralis major M.

Bilateral accessory axillary breast tissue in a 24 year old pregnant woman

⇒ **Polymastia (polymazia):**

- Multiple breasts may be present along the milk line (or even in thigh).
- Most common to occur in axilla.
- They may function during lactation

⇒ **Micromazia:**

- Small Breast
- TTT: augmentation mammoplasty.

⇒ **Macromazia (benign virginal hypertrophy):**

- Diffuse hypertrophy of the breast.
- Occur at puberty due to alteration of normal breast sensitivity to estrogen.
- TTT: reduction mammoplasty.

Idiopathic unilateral hypoplasia and contra lateral hyperplasia

There are 11 possible sites for polymazia on milk line on each side.

⇒ **Infantile gynecomastia:**

- Diffuse enlargement of male infant breast (unilateral or bilateral)
- Due to effect of circulating maternal sex hormones.
- Usually disappears in 6 months (requires no treatment)

Breast Trauma

- Blunt trauma to the breast produces 2 types of lesions that may be clinically difficult to distinguish from carcinoma; breast hematoma & traumatic fat necrosis.
- Sometimes, this trauma may draw the attention of the patient to an already existing

1. Breast Hematoma

- Following blunt trauma or breast surgery.
- If there is no external bruising, a deeply seated old hematoma may form a hard mass that greatly resembles breast carcinoma.
- Biopsy will confirm the diagnosis.

2. Traumatic Fat Necrosis

Pathogenesis

- Trauma → Fat Necrosis → Release of Fatty Acids → F.A. + Ca → Ca Soaps .
- This soap invites foreign body reaction.
- The result is one of two forms
 1. **A cyst** that contains thick oily fluid
 2. **A hard mass** (less frequent) that resembles breast carcinoma requiring biopsy to settle the diagnosis (cut section shows a characteristic chalky white appearance)

Clinical Picture

- History of trauma (**only in 50% of cases**).
- Hard, irregular, painless mass.

DD

- **Carcinoma** as this mass will be (**hard, dimpling** if it occurs near cooper's ligament, **retracted nipple** if near milk duct).

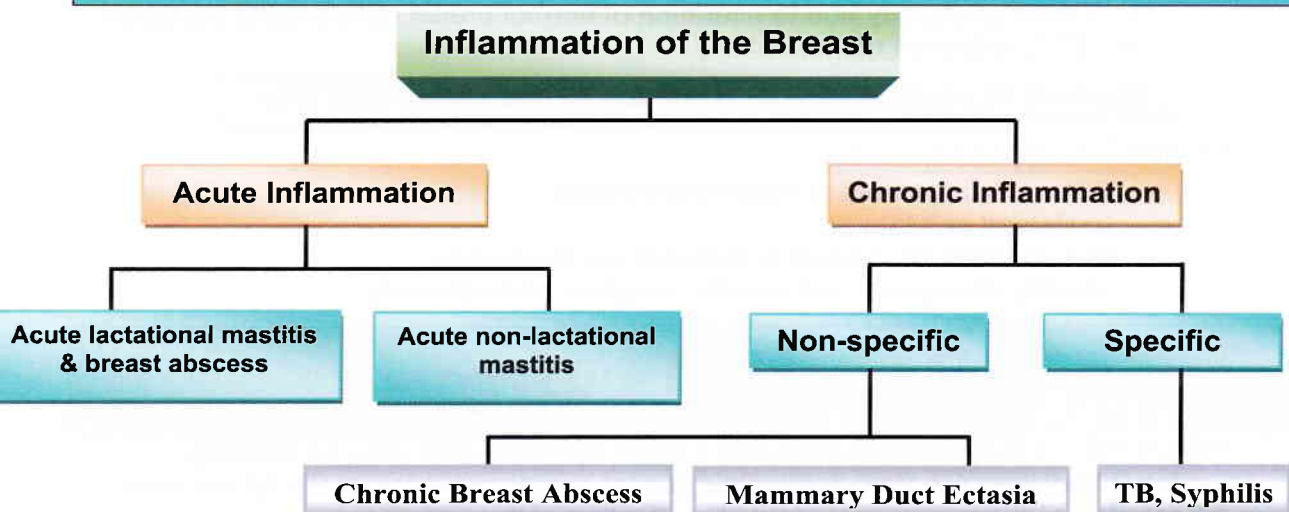
Investigations

- Biopsy → foamy fat laden macrophages. There is absence of yellow specks and gritty texture of carcinoma
- Mammography is not conclusive.

Treatment

- Excisional biopsy.

Inflammation of the Breast



Acute Inflammation

A. Acute Lactational Mastitis & Breast Abscess (most common mastitis)

Definition

- Acute bacterial inflammation of the breast which occurs during lactation.

Incidence

- Most common type of mastitis

Etiology

Causative Organism

- **Staph. aureus** (coagulase positive).
- The organism produces clotting of milk in ducts producing obstruction & stasis.

- **Etiology:** staph.aureus (direct, blood)
- **C/P:** 4 stages (engorgement, cellulitis, acute abscess, chronic abscess)
- **Comp:** general, local (sepsis)
- **Invest.:** sepsis + U/S
- **TTT:** prophylactic, curative (incision)

Route of Infection

- **Direct spread (Main route)** → from mouth of suckling infant through nipple cracks & opening of lactiferous duct.
- **Blood spread (less common)** from a septic focus.

Predisposing Factors

- Milk engorgement due to blockage of ducts by inspissated milk & epithelial debris.
- Nipple cracks & fissures caused by suckling
- Retracted nipple is more likely to be injured by the baby (who tries to get hold of it).
- Bad hygiene.
- Bad general condition as diabetes, steroid therapy.

Pathology

- At 1st infection is usually diffuse
- Milk engorgement → not treated well → acute mastitis → necrosis by staphylococci → multilocular abscess.

Clinical Picture

Milk engorgement → cellulitis → abscess → Chronicity

1. Stage of Milk Engorgement:

⇒ Symptoms:

- Dull aching pain.
- Mild persistent pyrexia.

⇒ Signs: enlargement & induration of the breast *with no signs of inflammation.*

2. Stage of Cellulitis (Mastitis):

⇒ Symptoms:

- The pain worsens
- Continuous high pyrexia

⇒ Signs:

- Diffuse redness, hotness & tenderness of the breast (signs of inflammation)
- Enlarged elastic tender LNs.

3. Stage of Acute Abscess:

⇒ Symptoms:

- Throbbing pain.
- Discharge (pus).

⇒ Signs:

- Hectic fever (i.e. at night it may reach 40° or more due to absorption of toxins due to vasodilatation).
- Edema of the overlying skin.
- No response to medical treatment. (Persistence of local signs > 5 days or severe systemic upset > 2 days after full antibiotic treatment)
- Fluctuation is a late sign.

(NEVER WAIT FOR FLUCTUATION IN BREAST ABSCESS)

4. Stage of Chronic Abscess: (See later)

- Attacks of remission & exacerbation
- Tender swelling with yielding center (**Paget's Test**).



Complications

- 1- General: toxemia, septicemia, pyemia.....
- 2- Local: chronicity.

DD

- 1- **Retro mammary abscess:** pain on pushing breast not on bimanual examination.
- 2- **Mastitis carcinomatosis:**

	Acute Lactational Mastitis	Mastitis Carcinomatosis
Symptoms:		
1. General	FAHM	Anorexia & loss of weight
2. Local	Pain of acute onset & rapidly progressive course	Pain of gradual onset & slowly progressive course
Signs:		
1. General	High fever	Low fever, cachexia
2. Local		
Signs of inflammation	+ve	-ve
Axillary LNs	Enlarged, tender, firm, mobile	Enlarged, not tender, hard, fixed
Skin	Rosy	Dusky red
Involvement	One breast sector is affected	More than 1/3 of breast
Fate:	The lesion either cured by Ab or forms an abscess	No response to Ab for 1 wk is an indication for biopsy

Investigations

(It's mainly a clinical diagnosis)

1. TLC, ESR & CRP → Increased.
2. Culture & sensitivity after drainage and to exclude mastitis carcinomatosis.
3. U/S → for detection of maturity of pus loculus and its location.

Treatment

⇒ **Prophylactic:**a) **During last 2 months of pregnancy:**

- Massage the nipple if not protruding.
- Good hygiene of nipple (Panthinol).

b) **After delivery:**

- Clean nipple after suckling by wet tissue (alcohol free).
- If the nipple is fissured → paint it with antiseptic cream.
- If milk engorgement occurs → Pump evacuation.

⇒ **Curative:****I. Before development of an abscess → Medical treatment**

⇒ Lactation should be stopped from the affected side with treatment according to stage :-

a) **MILK ENGORGEMENT:**

evacuate the breast with a breast pump in combination with hot backs

b) **CELLULITIS:**

as before with use of anti staph antibiotics (flucloxacillin or Augmentin ®) and analgesics, if the child is older than 9 months weaning should be advised

II. Stage of pyogenic abscess :

Surgical (incision and drainage):

Once pus loculus = drainage

Do not wait for fluctuation (late sign).

- I- General anesthesia.
- II- Incision:
 - a- Radial incision of the skin, not reaching areola
 - b- if small abscess → circum-areolar incision may be used for cosmetic purpose.
 - c- Counter incision might be needed to leave a drain (if the abscess is large & in a non dependant region).
- III- Introduce finger to destroy loculi and send pus for C & S.
- IV- Antibiotics & postoperative dressing (till healing is complete).
- V- Drain is removed when drainage stops

★ Some authors advise in cases of early breast abscess repeated aspiration guided by U/S under antibiotic cover and this often allows resolution without the need for an incision scar and will also allow the patient to carry on breast feeding.

Antibioma: if antibiotic is used in the presence of undrained pus antibioma will occur in the form of large sterile brawny edematous swelling that takes many weeks to resolve.

To Wean or Not to Wean

The advisability of weaning is controversial, but a reasonable approach is:

- 1- If baby > 9 months → stop feeding and give parodel 2.5 mg twice daily start with smaller dose then gradually increase.
- 2- If baby < 9 months → continue feeding with healthy breast and evacuation of diseased one by using pump.

B. Acute Non-lactational Mastitis

⇒ Mastitis Neonatorum:

- 1- It is due to withdrawal of mother estrogen.
- 2- Clinical Picture: Breast enlargement on 3rd or 4th day with breast discharge "Witch milk"
- 3- It is seen only in full developed infants.
- 4- **TTT:** leave it alone as it is self-limiting by 3rd week of life.

⇒ Mastitis of Puberty:

- 1. Painful tender enlarged breast (unilateral or bilateral)
- 2. Of both male & female at 14-15 years.
- 3. Self-limited within 2 years.

⇒ Traumatic Mastitis: due to ill fit brassiere.

⇒ Secondary infection of hematoma or tumor:

- Caused by anaerobes & treated by metronidazole 400 mg / 4 times daily for 5 days.

⇒ Pre mammary Abscess:

- Infection of Montgomery gland (apocrine, sweat gland of breast)
- **TTT:** incision & drainage.

⇒ Retro mammary Abscess:

- 1. Deep to pectoral fascia.
- 2. Due to **infected hematoma**, infected rib (osteomyelitis) or tuberculous empyema.
- 3. **Drainage:** by Thomas incision in the mammary groove.

Chronic Inflammation

A. Non-specific

Chronic Breast Abscess

Causes

- Inadequate treatment of acute abscess.
- Persistence of predisposing factors (i.e. Lactation).
- Bad General condition of the patient.

Pathology

- The abscess contains sterile pus.
- The organism present in the wall.
- Excess fibrosis.

- **Etiology:** bad (ttt + general condition + lactation)
- **C/P:** mass + pus + LNs
- **Invest.:** U/S + aspiration
- **TTT:** prophylactic + curative(excision)

Clinical Picture

- ⇒ **Type of patient:** history of maltreated abscess.
- ⇒ **Symptoms:**
 - Attacks of remission & exacerbation.
 - Breast mass: may be associated with nipple retraction or skin puckering
 - Pus discharge per nipple.
- ⇒ **Signs:**
 - Slightly tender breast swelling with yielding center (Paget test).
 - Axillary LNs. are enlarged elastic, tender & mobile.

DD

- 1) Fibro-adenoma.
- 2) Breast carcinoma.

Excess fibrosis makes the mass harder DD. → malignancy

	Chronic breast abscess	Carcinoma of breast
Symptoms:		
▪ History of acute abscess	+ve	-ve
▪ Purulent nipple discharge	May be present	Absent
Signs:		
▪ Fever	Low grade	Absent
▪ Surface	convex	Flat
▪ Tenderness	present	Absent
▪ Axillary LNs	Firm, mobile, discrete and tender.	Hard may be fixed & painless.
Investigations:		
▪ Aspiration	Reveals pus	Nothing
▪ Leucocytosis	Moderate	Absent

Investigations

- U/S (cyst) & needle aspiration (reveals pus and to exclude carcinoma).

Treatment

- ⇒ **Prophylactic Treatment:** adequate drainage of acute abscess.
- ⇒ **Curative Treatment:** Excision of the whole abscess.

Mammary Duct Ectasia

(Plasma Cell Mastitis)

(most common breast discharge)

Definition

- It is dilated **inflamed** major milk ducts.

Etiology

- Unknown.
- Theories:**

- Milk is a sequestered antigen, so in multipara milk may escape from milk ducts during lactation → Ag-Ab reaction → fibrosis → traction on the ducts → dilated ducts.
 - Anaerobic periductal infection followed by major duct dilatation
- There is a strong association between plasma cell mastitis and smoking.**

- **Etiology:** unknown
(Milk=AG, anaerobic infection)
- **C/P:** discharge + mass
- **D.D:** cancer breast
- **Invest.:** mass + discharge
- **TTT:** antibiotics + excision

Pathology

- Dilatation of major ducts which is filled with creamy secretion.
- Peri-ductal inflammatory reaction & fibrosis with plasma cell (Round cell) infiltration.
- Ductectasia is not a pre-cancerous condition

Clinical Picture

Type of patient

- Middle-aged female
- More common in **smokers**.

Symptoms

- May be **asymptomatic** or presenting by one of the following:
 - Nipple discharge:**

(Duct ectasia is the most common cause of nipple discharge)

- Arises from one or more ducts
 - May be creamy white, serous, yellowish or blood-stained
- subareolar Painless** or painful **swelling** if an abscess develops
 - Recurrent and chronic mastitis

Signs

- The affected area may be hard with skin dimpling & retraction of nipple (Fibrosis)



DD

- Breast carcinoma.

Investigations

If the patient presenting with subareolar mass, with or without nipple retraction

Triple assessment to exclude breast cancer (ductectasia shows coarse calcification in mammography)

If the patient presenting with nipple discharge

Benzidine test to exclude presence of blood
Cytological examination to exclude intraductal tumors

Treatment

- 1) Early & mild cases are treated by combination of antibiotic (flucloxacillin & metronidazole).
- 2) Correction of nipple inversion.
- 3) Persistent cases are treated by excision of major duct through circumareolar incision (Hadfield's operation).

B. Specific Breast Inflammation

1. TB Breast Abscess:

Incidence

- Rare with active pulmonary TB or 2^{ry} to cervical lymphadenitis.

Clinical Picture

- TB toxemia (night fever & sweat, Loss of appetite & weight).
- Multiple nodules in the breast or skin cold abscesses.
- Axillary nodes → enlarged and matted.

Investigation

- Biopsy → TB granuloma.

Treatment

- Anti-tuberculous drugs + simple mastectomy for resistant cases.

2. Syphilis:

- 1^{ry}: Chancre in nipple & areola + axillary lymphadenopathy.
- 2^{ry}: Condylomata lata + generalized lymphadenopathy.
- 3^{ry}: Gumma at skin.

Treatment

- Antisyphilitic drugs.

3. Actinomycosis:

- Rare with same character of fasciocervical actinomycosis.



Fibroadenosis

(most common breast disorder)

Other Names

- 1) **ANDI** → aberration in normal development & involution of breast.
- 2) Fibrocystic disease of the breast.
- 3) Sector mastitis.
- 4) Mammary dysplasia.
- 5) Chronic interstitial mastitis →
misnomer as in this condition there is no evidence of inflammation.

- **Etiology:** unknown (hormonal changes)
- **Path.:** fibrosis, adenosis, epith., cyst.
- **C/P:** cyclic (pain + mass + discharge)
- **Comp.:** anxiety + hge.&inf. + malignancy
- **Invest.:** mass + discharge
- **TTT:** mainly conservative

Definition

- Fibrocystic breast disease refers to benign (noncancerous) changes in the tissues of the breast. The term "disease" in this case is misleading, and many health care providers prefer the term "change."
- The condition is so common that it is believed to be a variation of normal..

Incidence

- The most frequent breast disorder (it occurs after puberty and before menopause).
- Most common between 30-50 years old

Etiology (Unknown)

1. The breast undergoes changes throughout the woman's reproductive life with cyclic changes during the menstrual cycle. The high prevalence of the disease in women in child bearing period points to a relation to ovarian cycle
2. **Other Factors**
 - a) High unopposed estrogen level with abnormal breast response
 - b) High Prolactin levels
 - c) Viral infection

Pathology

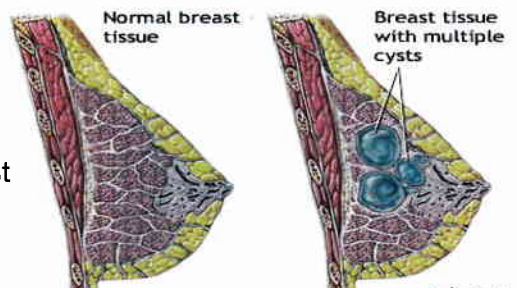
⇒ **The upper outer quadrant of the breast is the commonest site of affection.**

⇒ **It is Characterized by:**

1. **Adenosis** → Glandular hyperplasia with ↑ number acini.
2. **Fibrosis** → fibrous tissue replaces elastic & fatty tissue
3. Fibrosis → obstructs duct → retention cyst formation.
4. It may be unilateral or bilateral or affecting **sector** of breast.
5. **Epitheliosis** → Epithelial hyperplasia in small ducts.
6. Extensive epitheliosis → intra-ductal papillary growth which is termed **papillomatosis**
7. Rarely, there's "**Atypical epithelial hyperplasia**" → precancerous
8. Extensive fibrosis may resemble schirrous carcinoma & called "**sclerosing adenosis**"
9. Round cell infiltration.
10. **Cyst formation** → The cyst might be:
 - Small (microcyst).
 - Large macrocyst.
 - **The cysts may coalesce to form (blue domed cyst of Bloodgood):** A large cyst contains altered blood.

It is characterized by:

- Fibroadenosis.
- Fibrocystic dysplasia.
- Sector mastitis.



⇒ **It is NOT Precancerous:**

- **Most of the surgeons:**
 - Consider the fibroadenosis not precancerous.
 - Except if there is marked papillomatosis or atypical epithelial hyperplasia.

★ **Hyperplasia and papillomatosis increase risk of malignancy 1.5-2 times but atypical hyperplasia increase risk of malignancy 5 times**

Clinical Picture⇒ **Type of patient:**

- It occurs after puberty or before menopause, multiple painful lumps that may be unilateral or bilateral and it is related to menstrual cycle.

⇒ **Symptoms:**

- It may be completely **asymptomatic**.
- These changes are usually **CYCLIC**.

1- Breast pain (Mastalgia, Mastodynia):

- Exaggerated pre-menstrual tension
- Dull aching or stitching pain
- ↑ Pre-menstrually & by breast movement & ↓ post-menstrually & by breast support.
- Associated with enlargement and increase nodularity of breast

2- Breast lump (Cysts or sclerosing adenosis):

- The Most frequent complaint
- May disappear when the patient is re-examined 1 week after menstrual cycle

3- Breast discharge:

- Usually clear or yellow.
- Sometimes brown or green.

⇒ **Local Examination:****1- Breast Lump:**

- May be solid or cystic, freely mobile, commonly bilateral and diffuse.
- **Better to be felt by tip of fingers not by flat of the hand (painful nodularity).**

2- Discharge:

- With gentle squeeze.
- Colorless fluid or greenish discharge.

3- Axillary LNs:

- May be elastic, enlarged, tender and mobile with shotty distribution.

Non-cyclic Mastalgia

- It may be associated with ANDI or with periductal mastitis.
- It is more common in premenopausal females than postmenopausal.
- It should be differentiated from referred pain as musculoskeletal disorders.
- Breast pain in postmenopausal women not taking hormone replacement therapy is usually derived from chest wall.

Complications

1. If marked epitheliosis and papillomatosis increased risk of malignancy.
2. Hemorrhage and infection in cyst of Bloodgood.
3. Anxiety (if severe pain).

DD**1. Breast pain:**

1. Premenstrual tension.
2. Inflammation.
3. Malignancy (if advanced or mastitis carcinomatosis)

2. Breast lump:

1. Fibro-adenoma.
2. Breast carcinoma. (The most dangerous).

3. Breast discharge:

1. Duct ectasia (the commonest).
2. Fibroadenosis.
3. Duct papilloma (the commonest cause of bloody discharge).
4. Duct carcinoma.
5. Galactorrhea of pituitary adenoma.

Investigations

Investigations are usually not required but the following are indicated to rule-out breast cancer in suspected cases

For lump
(Triple assessment)

U/S & Mammography

Cystic

Aspiration (not blood stained aspirate, mass disappears completely, and does not recur within 2 weeks)

Solid

FNABC or open Biopsy if FNABC was not conclusive

For discharge

Cytological examination and benzidine test

Fibroadenosis

Normal

Treatment

Reassurance of the patient from cancer phobia is the most important

A. Change life-style:

1. Firm bra.
2. Avoid coffee, tea and chocolate.
3. Regular intake of 400 IU of vitamin E may be helpful.

B. Medical treatment:

1. Analgesic.
2. Regulation of the cycle.
3. Prim-rose oil single evening dose.
4. Parlodel 2.5 mg tab/twice /day (anti-prolactin).
5. Danazol tab!!! (last line of TTT as it causes acne & hirsutism)
6. Psychotherapy.

C. Indication of the surgery:

1. **Biopsy** → if doubtful diagnosis.
2. **Excision of the cyst** → large cyst (cyst of Bloodgood).
3. Cysts are treated by aspiration; recurring cysts, sclerosing adenosis or any cancer doubt are excised for biopsy.

Cases with atypical epithelial hyperplasia (discovered by biopsy) should be instructed to perform breast self examination monthly, physical examination every 3-6 months & mammography yearly.

Prognosis

- Exacerbations may occur at any time until the menopause except in patient receive hormonal replacement therapy.

Breast Neoplasm

Classification

Benign		Malignant	
Epithelial	Duct papilloma	Epithelial	Carcinoma
Mixed epithelial and mesenchymal	Fibroadenoma	Mesenchymal	Sarcoma Lymphoma

I- Benign Tumors of the Breast

Duct Papilloma

(Most common bleeding per nipple)

Incidence

- Usually in young woman (30-40 years).
- It may be Unilateral or bilateral.
- The most common cause of bleeding per nipple.

- **C/P:** discharge, mass, no LNs
- **Invest.:** discharge, mass, galactography
- **TTT:** Micro-dochectomy + Histopathology

Pathology

Site

- Usually situated in one of the main ducts near the nipple.

Macroscopic

- Single pedunculated mass that may ulcerate causing blood-stained discharge from the nipple.
- It may block the duct causing a retention cyst.

Microscopic

- Vascular connective tissue core + overlying hyperplastic epithelium.

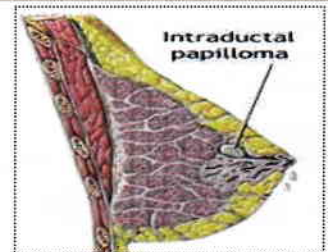
Clinical Picture

Type of Patient

- 30-40 years female with **bleeding per nipple**.

Symptoms

- Discharge:
 - Bloody or blood stained nipple discharge 50%. (Commonest symptom).
 - May be serosanguinous discharge.
- Swelling → retention cyst.



Signs

1. Bleeding per nipple:

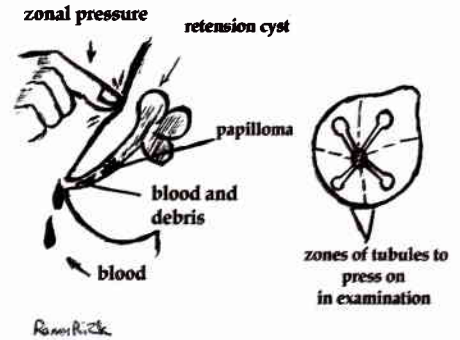
- By pressure on the swelling.
- If there is no palpable swelling, **zonal pressure** will reveal the discharge.

2. Swelling:

- Small, fusiform, usually lateral to the areola with its long axis pointing to the nipple.

3. Axillary LNs: are not enlarged.

No pain.



DD

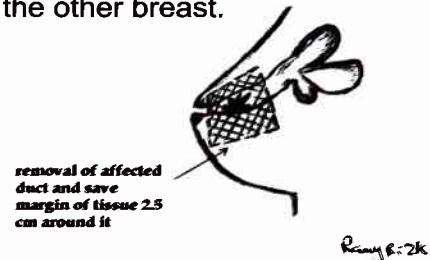
- Breast discharge, breast lump.

Investigations

- 1) **Benzidine test** → to make sure is it blood or not.
- 2) **Galactography** the papilloma appears as a regular filling defect.
- 3) **Mammography** → to screen the rest of the breast and the other breast.

Treatment

- It's a **pre-cancerous (10%)**, so the treatment is:
 1. **Micro-dochectomy** (remove the affected duct) through circumareolar incision and wedge of the tissue 2.5 cm around it.
 2. Histopathology.



How can you identify the affected duct intra-operatively?

- 1- By the lump.
- 2- If there is no lump, the duct is identified by passing needle through the discharging nipple opening.

Fibroadenoma

(most common breast mass in young)

Incidence

Fibroadenoma is the commonest cause of breast mass in young females

(The usual age is 15-30 years)

- **Path.:** fibrous, glandular
- **C/P:** painless (painful) lump
- **Invest.:** as mass
- **TTT:** hard: enucleation
Soft: excision

Pathology

1. Fibroadenoma is a **benign** neoplasm of the breast that affects both fibrous & glandular tissues but **fibrous element predominates**.

Types of Fibroadenoma

Peri-canalicular (Hard) (Benign simple)	Intra-canalicular (Soft) (Giant fibroadenoma)
Age: 20 - 30 years	Age: 30 - 50 years
Macroscopic picture: 1. Size: small 2. Surface: smooth. 3. Color: whitish 4. Consistency: firm or hard. 5. Cut section: whorly appearance. 6. Capsule: 2 capsules true and false capsule and a pedicle.	Macroscopic picture: 1. Size: large 2. Surface: lobulated. 3. Color: whitish 4. Consistency: soft 5. Cut section: might show central necrosis 6. Capsule: incomplete capsule.
Microscopic picture: - Formed mainly of fibrous tissue - Fibrous tissue proliferation occurs around the acini & ducts.	Microscopic picture: - Contains more glands - Fibrous tissue proliferation invaginates the ducts.
Complication: - Never turn malignant.	Complication: - Liable to turn to sarcoma.

Clinical Picture

I- Hard fibroadinoma

Type of patient 20 – 30 years aged female.

Symptoms Painless lump that is discovered accidentally

Signs Breast swelling:

- Usually small, non tender, firm, well-circumscribed with smooth surface & with **high mobility** in breast tissue (**breast mouse**). with no LN enlargement

II- Soft fibroadinoma

Type of patient 30-50 years old female

Symptoms painful rapidly growing lump



Intra-canalicular fibroadenoma

Signs Breast swelling

- May reach huge size, soft, mobile swelling in breast.
with no LN enlargement

DD

1. Breast carcinoma.
2. Duct ectasia.
3. Localized fibroadenosis.

Investigations

Clinical picture is usually enough for diagnosis

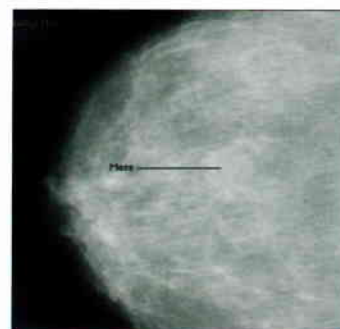
- Mammography → reveals well-circumscribed lesion.
- U/S → may be needed.

Treatment**1. For peri-canalicular:**

- It is **enucleated** through circum-areolar incision.

2. For intra-canalicular:

- If small, excision is better with a part of the normal breast tissue as a safety margin.



Fibroadenoma

Fibroadenosis	Fibroadenoma
Fibrosis +++	Fibrosis ++++++
Adenosis +++	Adenosis +
Disease of stroma and parenchyma	Disease of stroma with minimal parenchymal affection

- If large (Cystosarcoma phylloides) → wide local excision (to prevent recurrence) or if the tumor is occupying the whole breast → simple mastectomy.

Cystosarcoma Phylloides (serocystic disease of Brodie)

- **It is a type of soft fibroadenoma (giant intracanalicular fibroadenoma).**
characterized by:
 - 1- Highly cellular.
 - 2- Rapidly growing, painful and reaching a large size (20 – 30 cm).
 - 3- It might ulcerate through skin but not attached to it.
- **The name cystosarcoma phylloides (is a wrong name):**
 - Cyst: may be cystic degeneration if hugely enlarged due to insufficient blood supply (but usually it is not cystic)
 - Sarcoma: it is rarely malignant.
 - Phylloides: the cut surface resembles leaf.
 - So, it is better named "Phylloides Tumor"
- **Spectrum of activity:**
 - Variable from almost benign to locally aggressive & sometimes metastatic tumors.
- **Differential Diagnosis: carcinoma by probe test** passing a probe of glass between the tumor & skin:
 - If the probe can pass → benign.
 - If the probe cannot pass → malignant.
- **Treatment:** wide local excision to prevent recurrence or simple mastectomy if occupying the whole breast.

Massive swellings of the breast include:

- 1- Cystosarcoma phylloides.
- 2- Diffuse hypertrophy.
- 3- Giant fibroadenoma



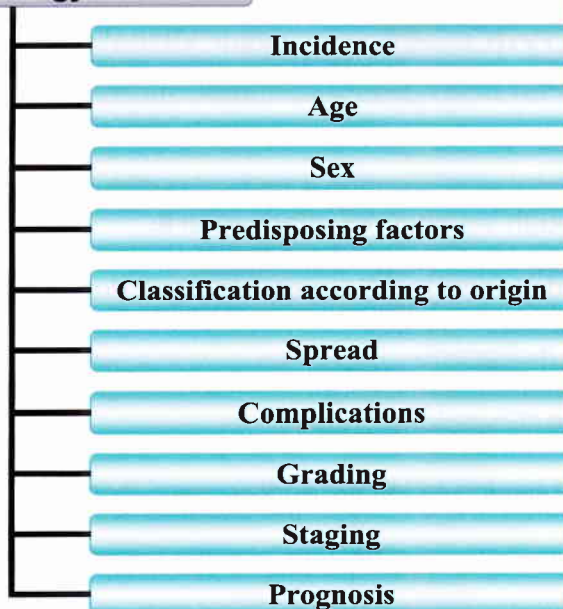
Cystosarcoma Phylloides

II- Malignant Tumors of the Breast

Breast Cancer

(most common cancer in Egyptian females)

Pathology



- **Types:** ductal, lobular, paget dis.
- **Spread:** direct, lymphatic, blood, transcelomic
- **Staging:** Manchester, TNM
- **C/P:** painless mass in upper outer quadrant
- **Invest.:** diag., staging, pre-op., follow up
- **TTT:** 1.**prophylactic:** early detection
2.**definitive:** early, late
3.TTT of complications

Incidence

- 1 in every 9 females in USA is expected to develop it.
- Breast cancer is the commonest malignancy in Egyptian females (35% of total malignancies).

Risk factors

Epidemiological data indicate well-defined factors increase the liability to develop the disease called relative risk (RR)

A. Major risk factors:

1. Age

- Carcinoma of breast is extremely rare below age of 20 years.
- Mean age of affection is 60 years.
- By the age of 90y nearly 20% of females are affected.

2. Sex

- 1) Breast cancer is 100 times more common in women than in men (RR 100)

3. Family history

- a) It has been proven that 5 - 10 % of breast cancers are due to **mutation in suppressor genes** (autosomal inheritance)

1. Mutation in 2 suppressor genes:

BRCA-I (on chromosome 17) & **BRCA-II** (On chromosome 13)

It usually occurs:

- At younger age
- Multifocal & bilateral
- 2. Mutation in tumor suppressor gene **P53**: producing **Li Fraumeni** \$
 - Breast cancer.
 - Ovarian cancer.
 - Carcinoma of the colon.
 - Lymphoma (or leukemia).

- b) **Positive family history** increases the risk:

- In mother or sister → ↑ the risk by 2.3 times.
- In **both** mother & sister → ↑ the risk 14 times.
- 20 % of breast cancers are familial

4. Previous affection with cancer breast

- Patient with breast cancer in one side, ↑ the risk to develop cancer in the other breast (cases of lobular carcinoma in situ RR 10).
- Bilateral breast cancer occurs in about 15 – 20 %.

(Up to 25 – 50 % if in lobular carcinoma)

5. Nulliparity

- Breast cancer is commoner in single & nulliparous women (RR 1.5)

6. Precancerous lesion

- 1) Duct papilloma (especially if multiple) → ↑ the risk 1.5 – 2 times.
- 2) Lobular carcinoma in situ → ↑ the risk by 5 – 10 times and it does not cause microcalcifications

B. Intermediate risk factors:

1. Age of menarche & menopause

- Early menarche (< 12 years) (RR 2.3).
- Late menopause (> 50 years)

2. Radiation therapy to the chest

- Women who had radiation therapy to the chest (including breasts) before age of 30 are at an increased risk of cancer breast e.g. Mantle radiotherapy in treatment of Hodgkin disease.

3. Obesity

- As there is peripheral conversion of steroid hormones into estradiol (E1) by aromatase enzyme in fatty tissues.

4. Benign breast diseases

- Atypical epithelial hyperplasia → ↑ the risk 2 - 5 times.

C. Minor risk factors

1. Alcoholic intake:

2. Oral contraceptive pills & hormonal replacement therapy (HRT):

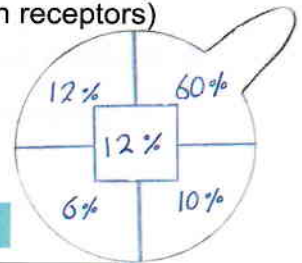
- Long term exposure to combined preparations of HRT does significantly ↑ risk of developing breast cancer.

3. Physical inactivity:

- Women who are physically inactive have an increased risk of breast cancer, because physical activities may help to reduce risk by preventing weight gain.

Site

1. Upper outer quadrant 60%. (due to ↑ breast mass & ↑ estrogen receptors)
2. Upper inner quadrant 12%.
3. Lower outer quadrant 10%.
4. Lower inner quadrant 6%.
5. Areola and nipple 12%.



Classification according to the origin

Duct carcinoma

Non-infiltrating (Duct carcinoma in situ)

Subtypes:

- 1- Comedo type.
- 2- Cribriform type 2%.
- 3- Micropapillary type
- 4- Papillary type 3%.
- 5- Solid type.

Lobular carcinoma

Infiltrating

Non-infiltrating (lobular carcinoma in situ) 1%

Subtypes:

- 1- Schirrous (not otherwise specified)
- 2- Medullary carcinoma
- 3- Mastitis carcinomatosis
- 4- Mucinous carcinoma (colloid)

Paget Disease of the nipple

Infiltrating 10%

- 25 %
- Bilateral
- Multi-centric

Subtypes of cancer breast

1- In situ Carcinoma 5%

- It is pre-invasive cancer that has not reached the epithelial basement membrane.
- It now accounts for over 20% of cancers detected by screening in UK.
- May be ductal (DCIS) or lobular (LCIS).
⇒ **LCIS** often bilateral and multifocal.

2- Schirrous carcinoma (not otherwise specified) 75%

- **The commonest histologic type of breast cancer.**
- Fibrous tissue is more prominent than malignant cells.
- **Macroscopic picture:** hard mass infiltrating edge + area of hemorrhage and necrosis and gritty sensation on cut surface and concave.
- **Microscopic picture:** malignant rounded cells and fibrous tissue.
- **Spread :** rapid lymphatic spread occurs through breast and there may be multiable satelli masses within the breast

3- Medullary carcinoma (encephaloid) 6%

- Malignant cells > fibrous tissue.
- **Macroscopic picture:** tumor cuts core soft in consistency like brain (so called encephaloid)
- **Cut surface** has central necrosis as if there is a cortex and medulla so called medullary carcinoma.
- **Microscopic picture:** malignant cells with little fibrous tissue + lymphocytic infiltration → good prognosis.

4- Mastitis Carcinomatosis (inflammatory carcinoma)

- **Occurs most commonly during pregnancy and lactation.**
- Is a rare and highly aggressive cancer
- Usually involves at least 1/3 of breast and may mimic a breast abscess.

5- Colloid Carcinoma 3%

- **Macroscopic picture:** tumor soft jelly-like with honey-comb appearance in formalin jar due to diluted mucin.
- **Microscopic picture:** spheroidal cells distended with mucoid material giving signet ring appearance.

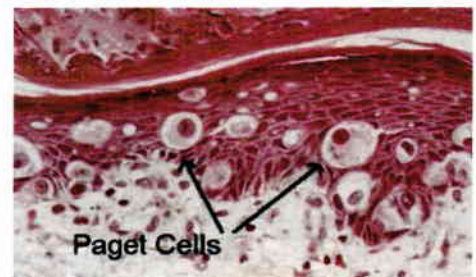
Mucinous carcinoma in breast has a good prognosis unlike mucinous carcinoma elsewhere in the body which usually has the worst prognosis.

6- Comedo Carcinoma

- Central necrosis of cancer cells → pasty blood tinged material on cut surface

7- Paget's Disease

- Intraductal carcinoma, begins in the epithelium of main milk duct and spreads within the epithelium up to skin of the nipple and down to breast substance
- **Macroscopic picture:** an eczema-like lesion and nipple is eroded
- **Microscopic picture:**
 - **Paget's cell:** large vacuolated cells, deeply stained nuclei (occur alone or in clusters).
 - **Hyperplasia of all layers of the epidermis.**
 - Lymphocytic infiltration → good prognosis.



Spread of Cancer Breast

A. Direct Spread:

1. Breast tissue.
2. Skin.
3. Pectoral fascia.
4. Pectoralis major.
5. Serratus anterior.
6. Chest wall.

B. Lymphatic Spread: (Commonest method of spread)

(By both embolization & permeation)

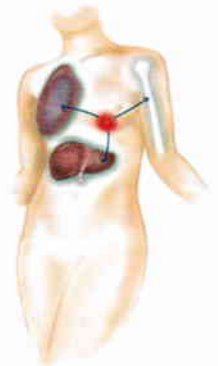
- Axillary LNs (common)
- Internal mammary LNs (next common)
- Supraclavicular LNs (in advanced disease)

- 1- Lymphatics from lower inner quadrant may pierce rectus sheath → liver nodules → falciform ligament → umbilical nodules (sister Joseph).
- 2- Obstruction of skin lymphatics causes edema of breast skin that is marked in dependent area giving the appearance of an orange peel; hence the French name "Peau d'orange".
- 3- Few lymphatics pierce the pectoralis major muscle to drain into the interpectoral LN of Rotter and pass along the intercostals bundles to reach the posterior intercostal LNs

C. Blood Stream Spread:

- Gives metastasis to **bone**, liver, lung & brain
- Bone secondaries are mainly **osteolytic** lesions
- The commonly affected bones by metastasis are **lumbar vertebrae**, femur, ribs & skull.
- The metastases to vertebrae are due to free (valveless) communication between posterior intercostals veins and paravertebral venous plexus.

Now it is well realized that cancer breast may spread by blood stream very early producing distant micrometastasis.



D. Transcelomic Spread:

(After liver affection)

1. Ovaries → **Krukenburg's tumor** (by retrograde lymphatic spread)
2. Douglas pouch → **Plummer's shelf nodules**
3. Peritoneum → **malignant ascites**.

Staging of Cancer Breast

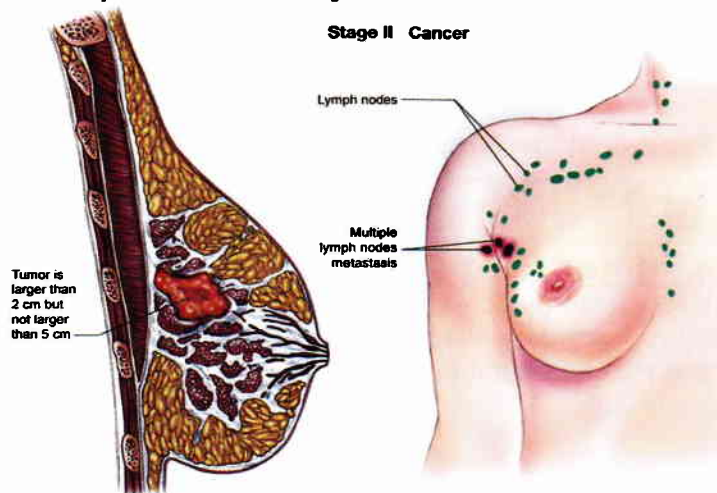
Manchester Staging (Better in Clinical)

Stage I

- Mobile mass in the breast.
- Not attached to pectoral muscles or chest wall.
- With or without skin tethering (in area **less** than tumor periphery).
- **No palpable axillary LNs.**

Stage II

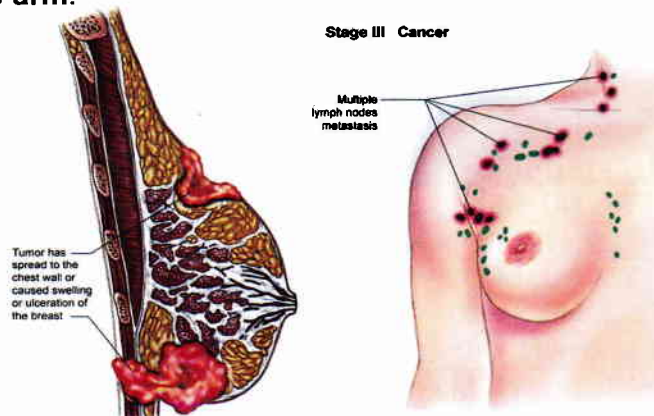
- Mobile mass in the breast
- Not attached to pectoral muscles or chest wall.
- With or without skin tethering (in area **less** than tumor periphery).
- **Palpable mobile ipsilateral axillary LNs.**



Stage III (stage of wide local spread)

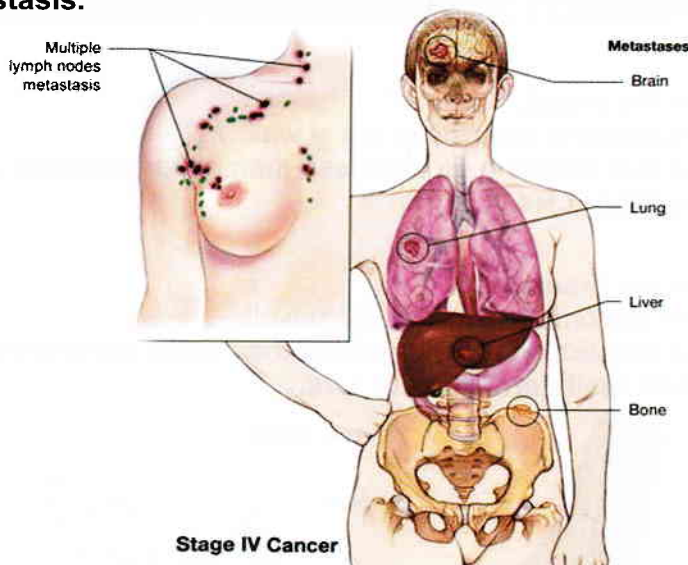
Any of the following:

- Skin affection (in area **more** than tumor periphery **but limited to breast**).
- **Fixed to pectoral muscles.**
- **Ipsilateral axillary LNs matted together.**
- **Ipsilateral supra clavicular LNs affection.**
- **Edema of the arm.**



Stage IV

- Skin affection **wide** of the breast (**Cancer en cuirasse**).
- **Fixed to chest wall**.
- Involvement of **opposite breast or axilla**.
- **Distant metastasis**.



International TNM classification (more accurate)

Tumor

T0 = not clinically felt (detected by screening).
 Tis = carcinoma in situ (detected by histopathology) or paget's disease with no palpable tumor.
 T1 = Tumor < 2 cm.
 T2 = Tumor 2-5 cm.
 T3 = Tumor > 5 cm.
 T4 = any size but fixed to the skin or chest wall or inflammatory carcinoma

NB:

Tx = tumor cannot assessed clinically as previous operation

Lymph Node

N0 = No palpable L.N.
 N1 = Mobile ipsilateral axillary LNs
 N2 = Fixed ipsilateral axillary LNs
 N3 = Palpable ipsilateral supra-clavicular LN or edema of the arm.

NB:

Nx = nodes cannot assessed clinically as previous operation

Metastasis

M0 = No evidence of distant metastasis.
 M1 = Distant metastasis
 Mx = Distant metastasis cannot be assessed

Staging of the UICC (union international contre cancer)

Stage UICC	Description	Category	5 year survival (%)
I	T1, N0, M0	Early breast cancer	93
II	IIA T2, N1, M0	Early breast cancer	72
	IIB T3, N0, M0		
III	IIIA T1-3, N0-2, M0	Locally advanced breast cancer	41
	IIIB T4, any N, M0		
IV	Any T, any N, M1	Metastatic	18

Clinical Picture of Cancer Breast

Symptoms

Female 50 – 60 years with painless swelling in upper lateral quadrant of the breast

1- Painless breast lump:

- Discovered **accidentally** (or during routine screening) and progressive course.

2- Less commonly:

- Watery (mostly) , Blood stained nipple discharge or pasty material.**
- Nipple retraction
- Breast pain in:
 - Late cases.
 - Mastitis carcinomatosa (inflammatory carcinoma)
 - Paget's disease of the nipple.

3- Menstrual history:

- Early menarche.
- Late menopause.
- Use of OCPs.

4- Past history:

- Cancer of the contra lateral breast.
- Irradiation to the breast.

5- Family history:

- Cancer breast in first degree relatives.

6- Occasional presentation:

- Bone → bone ache & pathological fractures.
- Lung → dry cough, hemoptysis & dyspnea.
- Liver → malignant jaundice and right hypochondrial pain.
- Brain (Rarely affected) → headache and mental changes and blurring of vision.
- Axillary LNs → axillary lump.

Signs

General

(*Cachexia + metastasis*)

- Cachexia and Troisier's sign (enlarged left supraclavicular lymph node).
- Liver:** jaundice, malignant ascites, hepatomegaly, sister Joseph nodules or peritoneal nodules (by PR or PV)
- Lung:** chest examination.
- Bone:** skull and spine examination.

Local

- **Both** breasts, axillae & supra-clavicular LNs must be examined.
- Examine the normal site first.

1. Inspection:

1- Breast enlargement & asymmetry.

2- Skin manifestations:

a. Nipple:

▪ Nipple Retraction:

- Due to infiltration of milk duct.
- **Not diagnostic** as it occurs in any fibrotic process e.g. chronic breast abscess & duct ectasia.

b. Areola: eroded in paget's disease of the nipple.



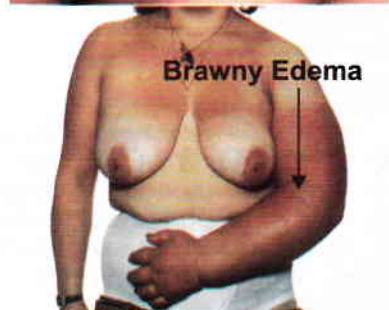
Nipple retraction



Paget's Disease

c. Skin proper:

- Skin tethering:
- Dimpling appears only in movement of breast as cancer infiltrates cooper's ligament but not to the degree of the appearance of dimpling.
- Skin Dimpling:
 - It's the **earliest** skin sign.
 - Due to contracture of **Cooper's ligament**
 - **Not diagnostic** as it occurs in any fibrotic process e.g. chronic breast abscess & duct ectasia.
- Skin Puckering.



Infiltration of cooper's ligament

Skin changes

Lymphedema

Metastasis

- Skin Nodules:
 - May appear away from mother carcinoma.
 - Due to retrograde lymphatic permeation.
 - **Diagnostic** (sure sign of malignancy).
- Skin Ulceration:
 - Can be differentiated from benign tumors by probing test.
- Cancer en Cuirasse:
 - Late stage of retrograde lymphatic permeation.
 - Skin is very **thick, leathery, brownish & metallic** simulating shields of war
- Peau d'orange:
 - Due to obstruction of lymphatics so lymphedema of skin occurs except at site of hair follicles & sweat glands.
- Brawny Edema (Lymphedema of the Arm):
 - ⇒ Due to:
 - Obstruction of lymph. Vessels by:
 - Tumor metastasis
 - Surgical.
 - Irradiation.
 - Obstruction of axillary vein.
- Sister Joseph Nodules:
 - Lymphatic spread to umbilicus.
- Special forms:
 - Mastitis carcinomatosa:
 - Skin is red, warm & edematous.

Brawny edema: Skin edema due to infiltration of lymph vessels.

Elephantiasis surgica: Skin edema due to surgical removal of L.N.s

2. Palpation: (start with examination of normal breast & axilla at first)

1- Breast Mass:

- Site → common in upper lateral quadrant (but may affect any site).
- Surface → irregular & flattened.
- Edge → ill defined.
- Consistency → hard (soft in medullary carcinoma).
- Mobility → restricted mobility within the breast.
Fixation to skin or muscle or chest wall is diagnostic of carcinoma



2- Lymph Nodes: (Bilaterally)

- Axillary LNs: if enlarged → hard, early mobile & late fixed.
- Supra-clavicular LNs.

3. Examination for possible metastasis (abdomen, chest,)

Differences between duct carcinoma in situ and lobular carcinoma in situ		
	DCIS	LCIS
Frequency	More common	Less common
Bilaterality and multicentricity	Rare	Common
Microcalcification	present	Absent
Early detection	Possible	Less likely
Potential for invasive cancer	30-50%	It is a marker of increased risk of malignancy in the same or other breast
Treatment	As invasive cancer	Strict follow up

	Early Breast Cancer = (T ₂ N ₁ M ₀) or stage I,II in Manchester	Late Breast Cancer = (>T ₂ M ₁ M ₀) or stage III, IV in Manchester
Symptoms	○ Painless swelling ○ negative occult presentations	○ Painful swelling ○ Positive occult presentations
General signs	Negative	May be positive for evidence of metastasis
Local signs:		
a- Inspection	1- Breast enlargement and asymmetry. 2- Skin lesions: a- Nipple retraction and skin dimpling. b- Peau de'range	1- Breast enlargement and asymmetry. 2- Skin lesions: a- Skin nodules. b- Sister joseph nodules. c- Cancer en cuirasse d- Skin ulceration. e- Brawny edema
b- Palpation	1- Firm to hard mass freely mobile. 2- Axillary LNs: negative or may be enlarged (hard and mobile)	1- hard fixed mass. 2- Axillary LNs: hard and fixed.

DD

A. Other causes of breast mass:

- | | |
|-------------------|---------------------------|
| 1- Fibro-adenoma | 2- Fibrocystic disease |
| 3- Breast cyst... | 4- Chronic breast abscess |
| 5- Duct ectasia | 6- Chronic fat necrosis. |

These 4 lesions constitute 95 % of breast lumps

	<i>Carcinoma</i>	<i>Fibroadenoma</i>	<i>Fibrocystic disease</i>	<i>Solitary cyst</i>
Age	Usually > 35y	15 – 30 y	35 – 55 y	35 – 55 y
Pain	Painless	Painless	May be painful	May be painful
Surface	Irregular	Smooth (may be lobulated)	Indistinct	Smooth
Consistency	Usually hard	Firm & highly mobile	Firm	Soft to hard
Axillary LNs	May be palpable axillary LNs	Free	Free	Free

B. Other causes of breast discharge.

C. Paget's disease & eczema:

Paget's Disease	Benign Eczema
Unilateral	Commonly bilateral
Commonly at menopause	Commonly at lactation
Starts in the nipple	Starts in the areola
Nipple is eroded	Nipple is intact
No itching	Itching
No vesicles – not oozing	Vesicles – oozing
No response to eczema treatment	Responds to eczema treatment
Well defined	Ill defined
A breast lump may be felt	No lump

D. Mastitis carcinomatosa & acute bacterial mastitis:

<i>Mastitis Carcinomatosa</i>	<i>Acute Bacterial Mastitis</i>
Affects more than 1/3 of the breast	Affects one sector of the breast
Gradual onset & slowly progressive	Acute onset & rapidly progressive
No or low grade fever	High fever
Skin is dusky red	Skin is rosy or firy red
Mild tenderness	Marked tenderness
Non-tender axillary LNs	Tender axillary LNs
No response to antibiotics in one week → Indicating biopsy	Responds either to antibiotics (or an abscess will be formed)

E. Nipple retraction

Investigations of Breast Cancer

Investigations

For Diagnosis

For staging

For pre-operative preparation

For follow up

- Taking a detailed family history is the first step in investigating a possible inherited predisposition to breast cancer.

1. For Diagnosis

1. **Soft tissue mammography:** (To evaluate the whole breast), 95% accuracy in diagnosis of breast cancer with expert hands

⇒ **Indications:**

- Screening for high risk group (main value).
- It is the only way to detect impalpable cancer breast.
- To evaluate the other breast in a patient with cancer breast.

⇒ **Finding in mammography suggestive of malignancy:**

1. **Clustered Micro-calcification:**

- These calcifications occur in ductal & not lobular carcinoma.
- 20% of micro-calcifications are malignant.

2. **Star shaped mass.**

⇒ **Useful in detection of multifocal lesion in the same or other breast**

2. **Ultra Sonography:** (Differentiate solid tumors from cystic)

⇒ **For cystic swelling we do: Aspiration**

The criteria of malignant cyst is

- Irregular wall.
- Hemorrhagic fluid.
- Rapid refilling.
- Residual mass after aspiration.
- Malignant cells in the aspirate.

⇒ **For solid swelling we do: FNABC**

- Mammography is of less diagnostic value in young women, in whom the density of lesion differs a little from normal tissue, so U/S in young women is useful
- Most authors nowadays: mammography is done with complementary U/S.

3. **Biopsy:**

- a) FNABC: simple, inexpensive and very accurate.
- b) Core-cut biopsy.
- c) Open biopsy.

4. **MRI of breast:**

- Gold standard of woman with synthetic prosthesis.

2. For Staging

1. **Sentinel Lymph Node Study:**

- Standard investigation in patients with clinically –ve LN affection.
- By injection of methylene blue or radioactive isotope → follow-up → till we find the sentinel lymph node.
- Then it is excised and frozen section is done for it to know whether affected by the cancer or not.

2. Lung → CXR.
3. Liver → abdominal ultra sound and liver function tests.
4. Bone → bone scan (Tc 99).
5. Brain → CT scan and MRI.

3. For Pre-operative Preparation

- CBC, FBS, LFTs, KFTs.

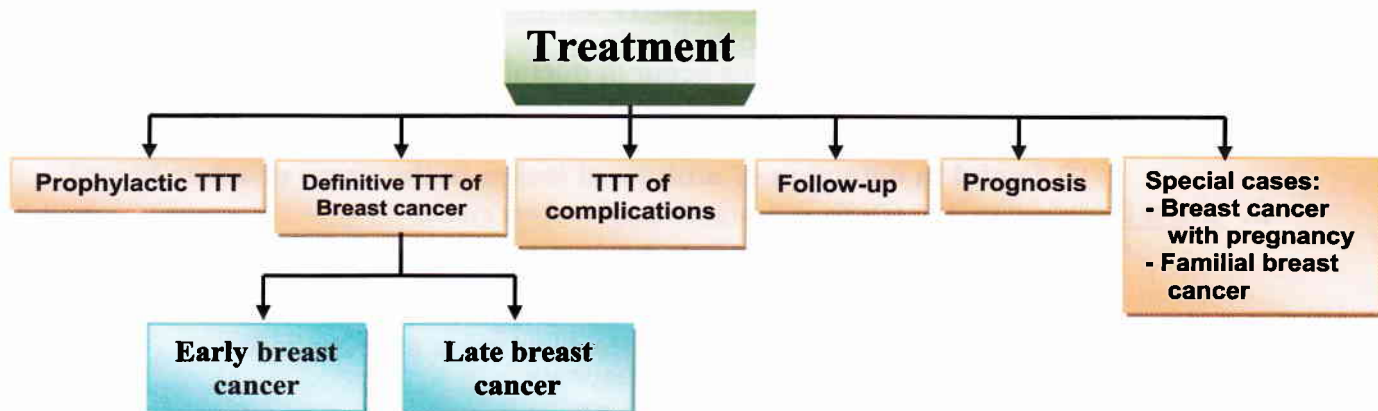
4. For Follow-up

- 1- **Tumor markers:** CA15-3 and CEA.

- 2- **Biochemical investigations (hormonal receptors):**

- About 60% of patients with breast cancers have estrogen receptors (ER- positive).
- Other breast cancers may have progesterone receptors.
- Staining for estrogen and progesterone receptors is now considered routine and their presence will indicate the use of adjuvant hormonal therapy with tamoxifen.
- Recently, tumors are stained for c-erb 52 (a growth factor receptor as patient can be treated with monoclonal antibody against this receptor to decrease relapse)

Treatment of Breast Cancer



Prophylactic Treatment

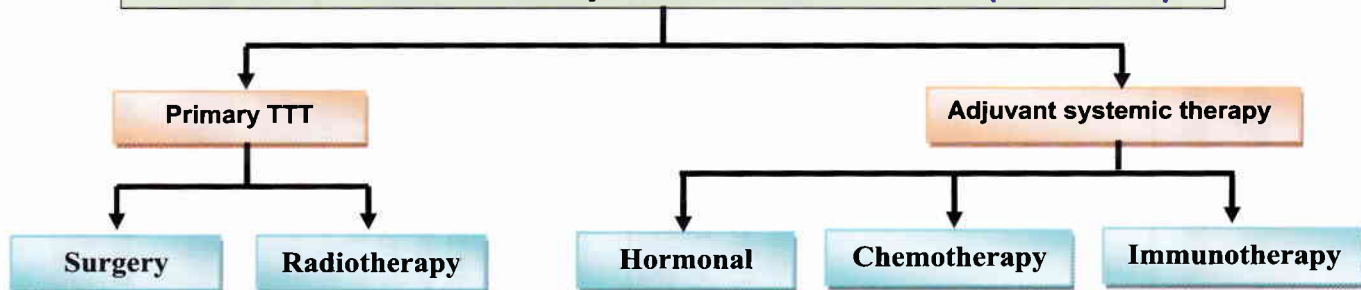
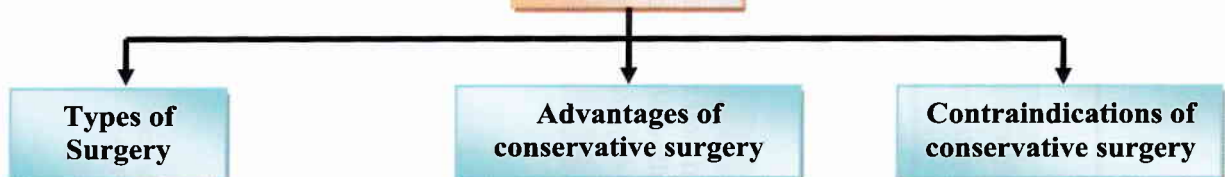
- There is no method to prevent breast cancer however, prognosis is markedly affected by early detection through the triad of:
 1. Self assessment.
 2. Physical examination.
 3. Mammography every 2 years.

Definitive Treatment of Breast Cancer

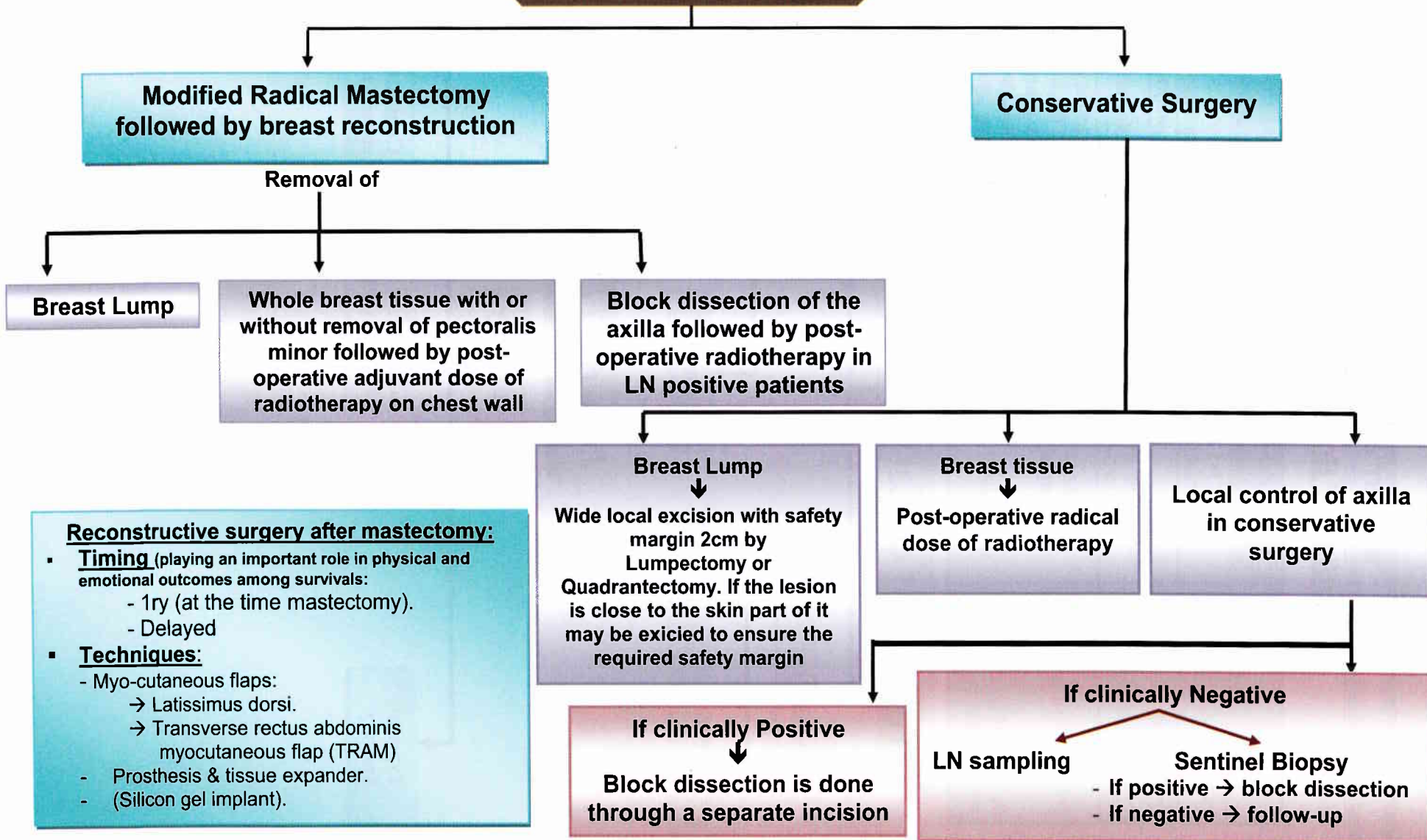
- Breast cancer is now widely accepted to be a systemic disease.
- In other words once it is evident clinically, it metastasize in the form of micrometastasis.
- Therefore, local and systemic treatments are indicated whatever the stage is.
- Basic principles for treatment of cancer breast:
 - Reduce the chance for local recurrence.
 - Reduce the risk for metastatic spread.

Outline of Breast Cancer Treatment

	Early breast cancer (Potentially curable)	Advanced breast cancer (Incurable)
Definition	T2 N1 M0 or less Manchester stage I or II	More than T2 N1 M0 Manchester stage III or IV
Aim	Cure	Palliation
Disease status	Mainly local disease ± micro-metastasis	Mainly systemic disease
Primary treatment	Surgery ± radio-therapy (Local treatment)	Chemotherapy & endocrinal therapy (Systemic treatment)
Adjuvant treatment (Palliative)	Endocrinal & chemotherapy	Simple mastectomy & radio- therapy have limited role in local control

Treatment of Early Cancer Breast (curable)**Primary TTT****1. surgery**

A- Types of Surgery



B. Advantages of conservative surgery over modified radical mastectomy:

1. Although local recurrence is high but the overall survival rate is the same.
2. Decrease psychological morbidity although recent studies show that 30% of the patients have anxiety or depression for fear of recurrence.

C. Contraindications of conservative surgery = indications of modified radical mastectomy: (MCQ)

I- Tumor:

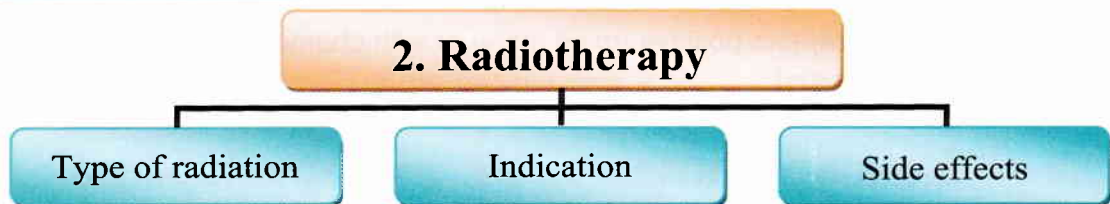
1. Bilateral & multi-focal disease.
2. Central lesions (surgery will cause bad cosmetic appearance).
3. Paget's disease of the nipple (bad cosmetic appearance, radio-resistant).
4. Tumor > 4 cm or small breast:
(we will need to remove additional 2 cm as a safety margin & therefore the cosmetic advantage of lumpectomy will not be achieved).
5. Distant metastasis.
6. Fixed to muscle.
7. High grade (**grade III**).
8. Insitu breast cancer more than 20 % due to the common incidence of multicentricity

II- Patient:

1. Pregnancy.
2. Patient preference.
3. Contraindication to irradiation e.g. SLE.
4. Previous irradiation.

III- Breast: relatively small in size.

Radiotherapy (Can be started after 14 days from surgery)



A. Type of Radiation:

- Deep X- ray (External beam).
- Ir¹⁹² wire implant (Interstitial Beam).

B. Indications:

1. Post-operative after conservative surgery to the remaining breast tissue by radical dose (5000 RAD).
2. Post-operative after radical mastectomy on the chest wall by adjuvant dose (1500 RAD) if:
 - a- High grade tumor or large tumor.
 - b- All LN positive patients.
 - c- Medial tumors for possibility of internal mammary LN affection.

C. Side Effects:

1. Local burn.
2. Interstitial pulmonary fibrosis.

⇒ **DCIS:**

May be treated by **Van Nuys system:**

Van Nuys System

- Non- high grade without necrosis.
- Non-high grade with necrosis.
- High grade.
- Van Nuys system is according to:
 - Patient age.
 - DCIS
 - Presence of micro-calcifications.
- Patient with high grade → benefit from radiotherapy after excision whereas of those of non high grade, who are completely excised → need no further treatment.

Adjuvant systemic treatment

A. Hormonal

- Indications
- Lines of TTT
- Results

B. Chemotherapy

- Indications
- Preparations
- Regimen

C. Immunotherapy

Hormonal Therapy

A. Indications:

1. If hormone receptors positive (used alone or with chemotherapy).

B. Lines of treatment:

Pre-menopausal	Post-menopausal
1- <u>First line of treatment:</u> tamoxifen 20 mg/day. 2- <u>Second line of treatment:</u> - Bilateral oophorectomy by: - a- Medical suppression (LHRH). - b- Surgery - c- Radiotherapy. - Adrenalectomy (rarely done) by : - a- Surgery. - b- Medical treatment by aminoglutethimide + cortisone.	<u>First line of treatment:</u> tamoxifen 20 mg/day. <u>Alternative hormonal agents:</u> 1- Aromatase inhibitors. 2- Raloxifene.

C. Results:

1. 60% improvement in estrogen receptor positive patients.
2. 80% improvement in progesterone receptor positive patients.

Chemo-therapy

A. Indications:

1. +ve LN biopsy (in pre-menopausal females)
2. – ve hormonal receptors.
3. High grade even if –ve LN or postmenopausal.
4. All patients below 70 yrs
5. Tumors more than 1 cm. } Kasr El-Einy book

B. Preparations (CMF):

- Cyclophosphamide.
- Methotrexate.
- 5-fluorouracil.

C. Regimen:

- Every cycle 8 days repeated every month for 6 months.
(Six cycles can reduce the risk of relapse 30%)

Target therapy

- For her2/neu receptor +ve cases → give monoclonal Ab (herceptin) against these receptors

More Details about Treatment

A. Hormonal therapy:

1- Tamoxifen (20 mg/day for 5 years):

- Mechanism of action: anti-estrogen (agonist-antagonist)
- Advantages:
 - a- Decrease annual rate of recurrence by 25% with 17% reduction in annual rate of death.
 - b- Effective in pre-menopausal and post-menopausal state.
 - c- Increase bone density.
- Side effects: hot flushes, increase risk of uterine cancer and thrombosis.

2- Aromatase inhibitors: (used post-menopausal only):

- Mechanism of action: inhibit aromatase enzyme.
- Advantages: no side effects of tamoxifen.
- Side effects: increase risk of osteoporosis.

3- Raloxifene: same as tamoxifen but with less side effects.

B. Prognosis:

- Presence of axillary LNs is the best marker for prognosis, however treatment of axillary LNs does not prolong survival rate suggesting that it does not act as a reservoir but as a marker for metastatic potential.

C. Radiotherapy

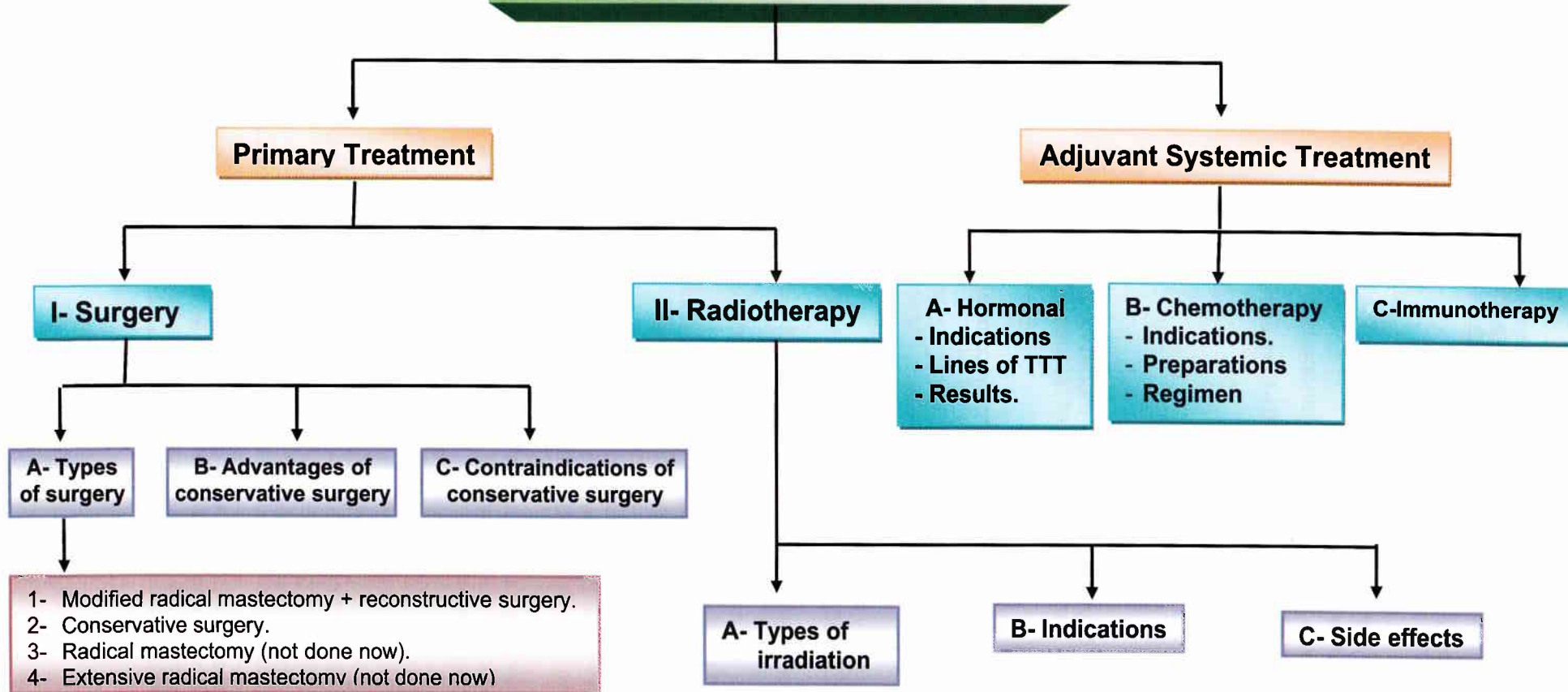
- Postoperative radiotherapy does not improve survival but it reduce the incidence of local recurrence (not started until the 14th postoperative day).

D. Chemotherapy

- CMF is no longer considered adequate adjuvant chemotherapy and modern regimen include anthracycline (epirubicin) and newer agents as taxanes.
- Adriamycin is commonly added in dose of 50mg/m²

Lymphangiosarcoma is a rare complication of lymphedema occurring years after treatment.
C/P: multiple subcutaneous nodules of upper limb, must be distinguished from recurrence.
Prognosis: bad but some cases may respond to cytotoxic therapy.

Treatment of early breast cancer



▪ Factors affecting the choice of treatment:

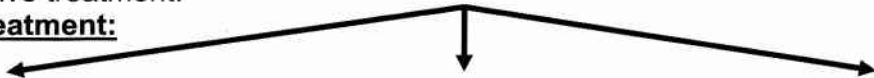
- 1- Stage and grade of the tumor.
- 2- Hormonal receptors.
- 3- Age and general health of the patient.
- 4- Menstrual state.

Intermediate (Locally advanced) breast cancer

- ➔ **This category of patients have:**
 - This category of patient have cancer above 5 cm diameter.
 - Fixed axillary L.N.s or internal mammary L.N.s.
- ➔ **For this:**
 - Distant metastasis should be excluded by C.T. scan & PET scan.
- ➔ **Treatment:**
 - Neoadjuvant chemotherapy → down staging:
 - If good response → breast conservative therapy.
 - If poor response → modified radical mastectomy.

Treatment of Advanced Breast Cancer (Incurable)

- **Aim:** palliative treatment.
- **Lines of treatment:**



A- Primary Systemic Treatment
 i- Hormonal therapy.
 ii- Chemotherapy.

B- Adjuvant Local Treatment
 i- Radiotherapy.
 ii- Palliative surgery

C- Treatment of Complications

A- Primary Treatment

i- Hormonal Therapy:

- a. Only given to hormone receptor +ve patient with 60-70 % response
- b. More effective in post-menopausal women
- c. Not very effective in hormone negative patients (response 10%), with visceral metastasis or young patient below 35 years old
- d. Tamoxifen (anti-estrogen) is given for not more than five years to avoid the risk of endometrial cancer or thrombogenicity. If no response aromatase inhibitors may be used

ii- Chemo-therapy:

- **Indications:**
 1. Rapidly progressive disease
 2. Premenopausal women
 3. Visceral metastasis
 4. Hormonal receptor –ve cases.
- **Combination:**
 - Cyclophosphamide, methotrexate & 5-fluorouracil (CMF).
 - Adriamycin is commonly added.

B- Adjuvant Treatment

i- Radio-therapy: It has a palliative role in the following situations:

- 1- Pain (due to bone or soft tissue involvement).
- 2- SVC obstruction.
- 3- used to control tumor fungation

ii- Surgery:

1. Modified radical mastectomy in stage III.
2. Palliative simple mastectomy in stage IV to get rid of unpleasant fungating tumor.

C- Treatment of Complications

1. Hypercalcemia:

- Correction of dehydration by IV fluids + furosemide
- Prednisolone + biphosphonates

2. Pathological fractures:

- Immobilization + internal fixation
- Radiotherapy to the fracture site.

3. **Cerebral metastasis**
 - Corticosteroids and radiotherapy
4. **Spinal cord compression:**
 - Surgical cord decompression with stabilization followed by radiotherapy
5. **Superior vena cava obstruction:**
 - Radiotherapy is the treatment of choice
6. **Pleural effusion:**
 - Systemic therapy and chest tube drainage
7. **Liver metastasis:**
 - Treated by chemotherapy.
8. **Lymphedema:**
 - Can be treated by complete decongestive therapy.

Post-operative radiotherapy does not improve the survival but it reduces the incidence of local recurrence.

Follow-up of Patients with Breast Cancer

⇒ After treatment, every 3 months for first 2 yrs, then every 4 months next 2 yrs, then yearly for life. to:

- 1- **Detect and treat complications of mastectomy**
 - Psychiatric morbidity caused by the loss of the breast
 - Arm edema results from excision of lymphatics, their obstruction by radiotherapy, lymphangitis caused by infection, or malignant axillary recurrence blocking them. Thrombosis of the axillary vein.
 - Avoidance of radiotherapy to the axilla which has been surgically evacuated of its nodes reduces the possibility of lymphatics edema.
 - The patient is warned to avoid minor trauma to the ipsilateral hand and should wear gloves when carrying out rough work. Arm elevation, massage, and elastic or pneumatic arm compression are partially effective.
- 2- **Detect local recurrence or distant disease:** because of the incidence of cancer in the other breast (1% per year) annual mammography of the contralateral breast is done.
- 3- **Instructions.** Patients are instructed not to get pregnant for at least three years, and to use non-hormonal contraception, to avoid the stimulating effect of hormones on possible residual tumor

Summary of Active Treatment of Cancer Breast

Early Breast Cancer

⇒ **Primary Treatment**

A. Surgery:

- Types of surgery

- 1- **Modified radical mastectomy + reconstructive surgery: Removal of:**
 - a- Breast Lump
 - b- Whole breast tissue with or without removal of pectoralis minor followed by post-operative adjuvant dose of radiotherapy on chest wall
 - c- Block dissection of the axilla followed by post-operative radiotherapy in LN positive patients
- 2- **Conservative surgery: in which**
 - a- Removal of Breast Lump by Wide local excision by Lumpectomy or Quadrantectomy.
 - b- Post-operative radical dose of radiotherapy for Breast tissue
 - c- Local control of axilla if clinically +ve or with sampling by block dissection through a separate incision.

B. Radiotherapy:

- ✓ Post-operative after conservative surgery to the remaining breast tissue by radical dose (5000 RAD).
- ✓ Post-operative after radical mastectomy on the chest wall by adjuvant dose (1500 RAD)

⇒ **Adjuvant Systemic Treatment**

A. Hormonal therapy: if hormone receptors positive

B. Chemotherapy: by (CMF) {cyclophosphamide, methotrexate & 5-fluorouracil}

- **Regimen:** Every cycle 8 days repeated every month for 6 months.

- **Indication:**

- +ve histological involvement of removed axillary nodes
- -ve hormonal receptors

C. Immunotherapy: recent trends.

Late Cancer Breast⇒ **Primary Treatment**

A. Hormonal therapy: if hormone receptors positive or bone metastasis.

B. Chemotherapy: by combination of CMF + Adriamycin

⇒ **Adjuvant Treatment**

A. Radiotherapy: in cases of pain or SVC obstruction

B. Surgery: palliative simple mastectomy

NB. Follow up of the patient for metastasis**Breast Cancer with Pregnancy**

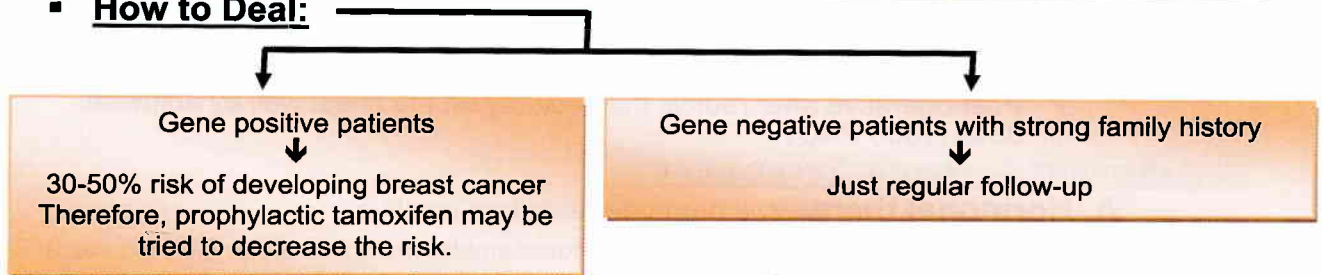
- The effect of pregnancy on breast cancer is not well-understood but if breast cancer develops in pregnancy, it tends to be at a later stage because it is masked by symptoms of pregnancy and lactation. (also may be due to increase vascularity)
- **Treatment:**
 - 1- Mastectomy is more optional than conservative surgery.
 - 2- Radiotherapy is contraindicated.
 - 3- Chemotherapy is not give in the 1st trimester.
 - 4- Hormonal treatment is usually not given because most of the tumors are with -ve hormonal receptors.

Familial Breast Cancer

- **Incidence:** less than 5%.
- **Genes involved:**
 - a- BRCA-1: in chromosome 17, associated with ovarian cancer (50%)
 - b- BRCA-2: on chromosome 13, associated with male breast cancer.
 - c- P53
- **The important features to look for in a family history are:**
 - Age at onset.
 - Bilateral disease.
 - Male breast cancer.
 - Multiple cases in one side of the family.
 - Ovarian cancer.

The great majority of women with a family history of breast cancer does not fall into a high risk group and do not develop breast cancer.

▪ How to Deal:



Prognostic Factors of Breast Cancer

- Type of the tumour.** The best prognosis is provided by the in situ carcinoma, and paget's disease, while the worst is the inflammatory carcinoma
- The T stage** of the primary tumour. The higher the T stage, the worse is the prognosis.
- Size, mobility, number, and location of the involved lymph nodes.**
 - Large fixed nodes are of bad prognosis
 - The number of involved nodes largely affects the prognosis
 - Patients with negative axillary nodes have a 10 years survival rate 65%
 - Patients with 1-3 positive axillary nodes have a 10 years survival rate of 38%
 - Patients with more than 4 +ve axillary nodes have a 10 years survival rate of 13%
 - The prognosis worsens the higher the affected nodes in the axilla. Involvement of level III nodes carries a bad prognosis
- The presence of distant metastasis markedly worsens the prognosis
- Hormone receptor status
- The site of the tumour. Medial half tumour have a worse prognosis than those of lateral half due to early involvement of the internal mammary lymph nodes

Diseases of Male Breast

Cancer Breast in Male

- **Incidence:**
 - It's a rare disease, the incidence is only about 1% of that of women
 - There may be an increase incidence of breast cancer in men with prostatic cancer in addition BRACA-2 mutation are common in men with breast cancer.
- **Clinical findings:**
 - Painless lump beneath the areola at 50 years.
 - Nipple discharge or retraction or ulceration.
- **Spread:**
 - Rapid spread to skin and chest wall due to deficient breast tissue
 - Blood born metastases are common.
- **DD:** gynecomastia & metastasis from other tumors.
- **Treatment:**
 - Like cancer breast of female.
 - **NB:** castration in advanced breast cancer is a successful measure and more benefital than the same procedure in women but is rarely used.
- **Prognosis:**
 - The prognosis of breast cancer is poor in men than in women.

Gynecomastia

Definition

- It's painless enlargement of male breast due to increased glandular elements ≠ Lipomastia.

Incidence

- About 65 percent of 14-year-old boys have gynecomastia; the condition often will improve after two to three years without intervention.
- Infants may have a transient form of the disease that is often related to the maternal hormones.

Etiology

- Idiopathic.** (the commonest cause)
- Physiological:**
 - Neonatal → from exposure to high maternal estrogen.
 - Pubertal → resolves in 2 years.
 - Old age → ↓ testicular function.
- Pathological:**
 - ↑ Estrogen:**
 - Feminizing tumors of testis (Sertoli cell tumor).
 - Feminizing tumors of adrenals.
 - Paramalignant syndrome as bronchogenic carcinoma.
 - ↓ Testosterone:**
 - Orchidectomy.
 - Testicular atrophy: mumps, leprosy and heat exposure.
 - ↓ Metabolism of estrogen, liver cell failure.**
- Iatrogenic:**
 - Digitalis.
 - Aldactone.
 - Reserpine.
 - Cimetidine.
 - Estrogen therapy as in cancer prostate.
- Genetic:** Klinefelter syndrome.

Clinical Picture

- History of drug intake.
- Abdomen → hepato-splenomegaly
- Testis → atrophy

Grading of Gynecomastia

- Grade I gynecomastia:** a small amount of increase in breast tissue is present with no extra skin.
- Grade II gynecomastia:** a moderate amount of enlargement of the breast can be seen with or without extra skin.
- Grade III gynecomastia:** an exceptional enlargement of the breast with extra skin.

- **Etiology:** idiopathic, physiological, pathological, iatrogenic, genetic
- **C/P:** bilat.(unilat.) breast enlargement
O/E: tender disc
- **Grading:** 3 grades
- **Invest.:** cause + mammogram
- **TTT:** reassurance. ttt of the cause



Investigations

1. **Blood tests** (including liver function tests and hormone studies)
2. **Urine tests**
3. **Consultation with an endocrinologist** : a physician who specializes in the functioning of hormones and how the hormones affect multiple organs.
4. **Mammogram** : a low-dose x-ray of the breast.

Treatment

A- If 2ry:

TTT of the cause.

B- If 1ry: (most cases require no treatment)

1. **Subcutaneous mastectomy:**
2. **Suction lipectomy:**
This is a form of liposuction that allows for tapering of the edges of the tissue without unwanted side effects.
3. **Endoscopic surgery:**
 - This newer procedure uses a small, flexible tube with a light and a camera lens at the end (endoscope) to examine the inside of the breast.
 - Tissue is then removed without placing a large, open, surgical incision.

Early Detection of Cancer Breast

Definition

- Finding cancer before it starts to cause symptoms.

Benefit

- Makes the treatment more successful.

Early Signs

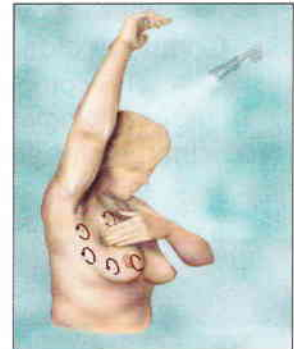
1. A lump → single, firm, and most often painless.
2. Unusual appearance of → the skin on the breast, underarm.
3. Veins on the skin surface become more prominent on one breast.
4. Inverted nipple develops a rash or has a discharge other than breast milk.
5. A depression on the breast surface

Detection Plan

- Clinical breast examinations (CBE) every 3 years from ages 20-39, then every year thereafter.
- Monthly self-breast examinations (SBE) beginning at age 20. Look for any changes in your breasts.

⇒ **How to do a Breast Self-Examination:**

- **IN THE SHOWER** Fingers flat, move gently over every part of each breast. Use your right hand to examine left breast, left hand for right breast.
- Check for any lump, hard knot or thickening. Carefully observe any changes in your breasts.
- **BEFORE A MIRROR** inspect your breasts with arms at your sides. Next, raise your arms high overhead.
- Look for any changes in contour of each breast, a swelling, a dimpling of skin or changes in the nipple. Then rest palm on hips and press firmly to flex your chest muscles. Left and right breasts will not exactly match - few women's breasts do
- **LYING DOWN** Place pillow under right shoulder, right arm behind your head. With fingers of left hand flat, press right breast gently in small circular motions, moving vertically or in a circular pattern covering the entire breast. Use light, medium and firm pressure. Squeeze nipple; check for discharge and lumps. Repeat these steps for your left breast.



- **Mammography:**
 - Baseline mammogram by the age of 40.
 - Mammogram every one to two years for women 40-49, depending on previous findings.
 - Mammogram every year for women 50 and older.
 - A personal calendar to record your self-exams, mammograms, and doctor appointments.
- **Breast U/S**
 - Sometimes used to evaluate breast problems found during screening or diagnostic mammogram or on physical examination
 - Used in addition to mammography not instead of it.
- **MRI:**
 - Not recommended in early detection of cancer breast as it is expensive and time consuming
 - Used with mammogram to detect cancer breast especially in women with very dense breasts
- **Full-filled digital mammogram (FFDM):**
 - It differs from standard mammogram as in FFDM the films are recorded and viewed by computer not on photographic films.
- **Computer aided detection and diagnosis (CAD):**
 - The computer will scan the mammogram at first then find tumor that the radiologist can not spot.
- **Scintimammography:**
 - Injection of radio-active tracer into a vein to detect breast cancer cells by special cameras.

Oral Questions

Paget's disease of the Nipple

Incidence

- 1 % of cancer breast **affects middle-aged and elderly women.**

Etiology

- It may be due to:
 1. Intraductal carcinoma (from ducts inside nipple and areola).
 2. Retrograde lymphatic spread from schirrous carcinoma.

Pathology

Gross Picture

1. It presents as an eczema-like condition of nipple and areola.
2. Nipple is eroded slowly

Microscopic Picture

- 3 characteristic features:
 1. Paget cells: large vaculated cells, deeply stained nuclei.
 2. Hyperplasia of all layers of the epidermis.
 3. Lymphocytic infiltration → good prognosis.



Clinical Picture

- 1- Pricking sensation of nipple.
- 2- Superficial erosion of nipple.
- 3- At early stages a mass may not be palpable & may appear only after 2 years.

Old female presented with unilateral eczema resistant to treatment

DD

Paget's Disease	Benign Eczema
Unilateral Commonly at menopause Starts in the nipple Nipple is eroded No itching No vesicles – not oozing No response to eczema treatment Well defined A breast lump may be felt	Commonly bilateral Commonly at lactation Starts in the areola Nipple is intact Itching Vesicles – oozing Responds to eczema treatment Ill defined No lump

Investigations

- Biopsy and histopathology.

Treatment

- It's considered cancer breast stage 1:
 - Modified radical mastectomy
 - It is radio-resistant.

Prognosis

- Good.



Sir James Paget is considered one of the founders of modern pathology. He was born in Great Yarmouth the 8th of 16 siblings. His father was a brewer, ship-owner, Chandler and sometimes mayor. His elder brother George Edward Paget (1809-1892) became Regius professor at the University of Cambridge.

Sarcoma

- Very rare.
- It occurs at 30 - 40 years
- It may be denovo or on top of soft fibroadenoma.
- It forms a fleshy rapidly growing massive tumor.
- Often huge with cystic degeneration & necrosis.
- Metastasis (**mainly blood** but **LN spread is rare & late**).
- It may be round or spindle cell sarcoma.
- **DD:** from medullary carcinoma.
- **Treatment:**
 - Palliative simple mastectomy (as there is no LN affection).
 - Radiotherapy.
- **Bad prognosis.**

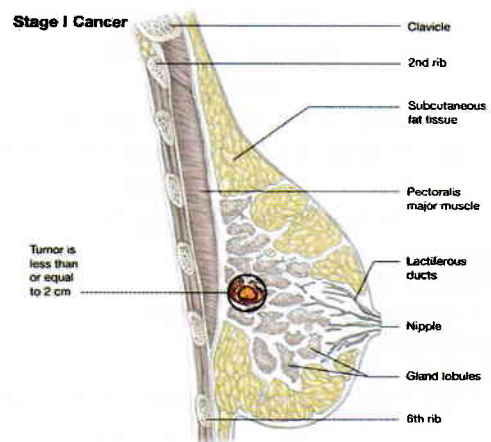
Other Methods of Staging of Breast Cancer

■ Stage 0:

- Ductal carcinoma in situ is cancer that has not spread past the ducts or lobules of the breast (the natural boundaries). It is also called non-invasive cancer.

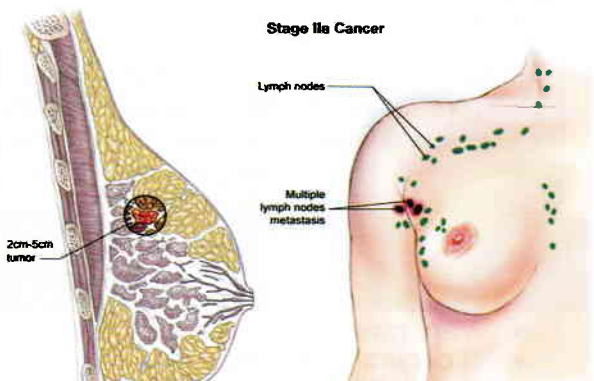
■ Stage I:

- The tumor is small and has not spread to the lymph nodes (T1, N0, M0)



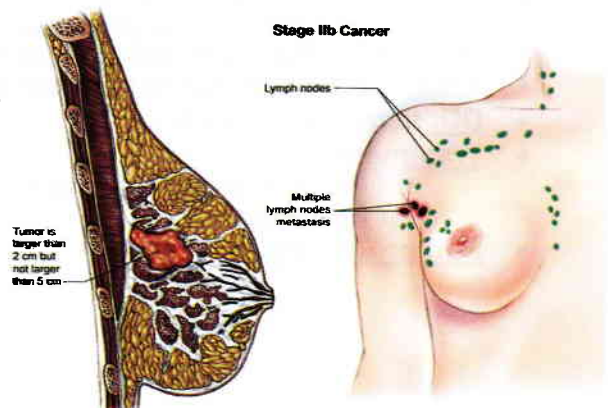
■ Stage IIa:

- Any one of these conditions:
- The tumor is ≤ 2 cm and has spread to the axillary lymph nodes under the arm (T1 or T1mic, N1, M0).
- The tumor is 2-5 cm and has not spread to the axillary lymph nodes (T2, N0, and M0).
- There is no evidence of a tumor in the breast, but there is cancer in the axillary lymph nodes (T0, N1, and M0).



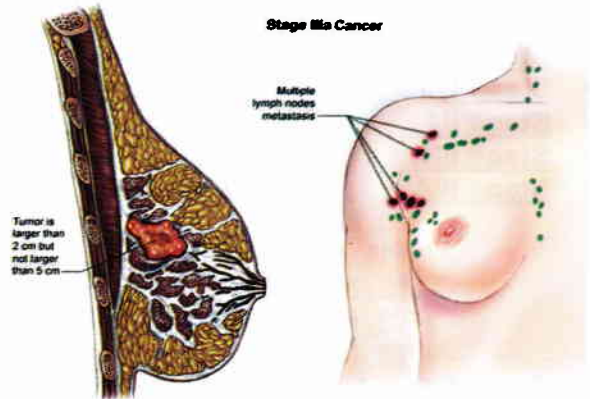
■ Stage IIb:

- Any one of these conditions:
- The tumor is larger than 2 cm but not larger than 5 cm and has spread to the axillary lymph nodes (T2, N1, M0).
- The tumor is larger than 5 cm but has not spread to the axillary lymph nodes (T3, N0, M0)



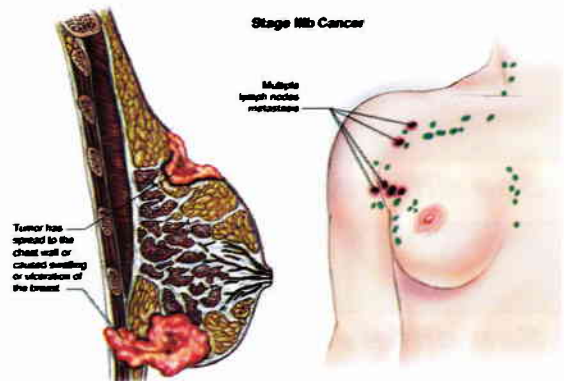
■ **Stage IIIa:**

- Any of these conditions:
- The tumor is smaller than 5 cm and has spread to the axillary lymph nodes (T1, N2, M0 or T2, N2, M0).
- The tumor is larger than 5 cm and has spread to the axillary lymph nodes (T3, N1, M0 or T3, N2, M0)



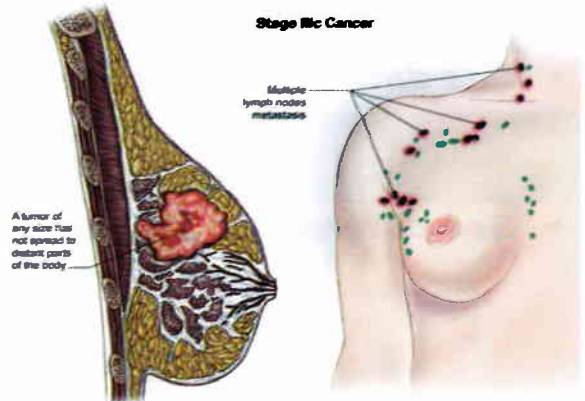
■ **Stage IIIb:**

- The tumor has spread to the chest wall or caused swelling or ulceration of the breast or is diagnosed as inflammatory breast cancer. It may or may not have spread to the lymph nodes under the arm, but has not spread to other parts of the body (T4, N0, M0; T4, N1, M0; or T4, N2, M0)



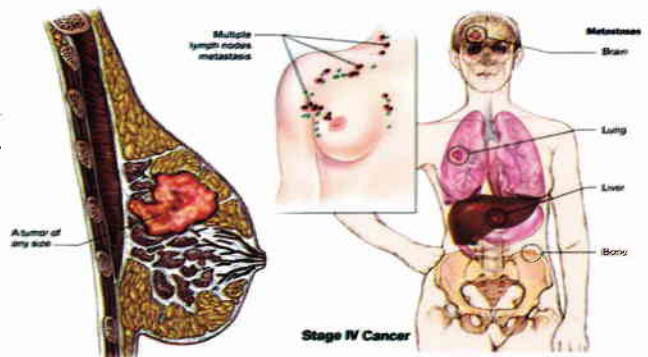
■ **Stage IIIc:**

- A tumor of any size has not spread to distant parts of the body but has spread to the lymph nodes in the N3 group (any T, N3, M0)



■ **Stage IV:**

- The tumor can be any size and has spread to distant sites in the body, usually the bones, lungs or liver, or chest wall (any T, any N, M1).



AJCC Staging

- **Stage 0:** Tis, N0, M0
- **Stage I:** T1*, N0, M0
- **Stage IIA:** - T0, N1, M0 - T1*, N1, M0 - T2, N0, M0
- **Stage IIB:** - T2, N1, M0 - T3, N0, M0
- **Stage IIIA**
 - T0, N2, M0
 - T1*, N2, M0
 - T2, N2, M0
 - T3, N1, M0
 - T3, N2, M0
- **Stage IIIB:** T4, N0, M0 T4, N1, M0 T4, N2, M0
- **Stage IIIC:** Any T, N3, M0
- **Stage IV:** Any T, Any N, M1

Cysts of the Breast

- ***See differential diagnosis book***

Management of Breast Lump

- ***See differential diagnosis book***

Management of Breast Pain

- ***See differential diagnosis book***

Management of Nipple Discharge

- ***See differential diagnosis book***

CHAPTER 2

THE THYROID GLAND

Anatomy

➤ See surgical anatomy book.

Congenital Anomalies

1. Congenital Aplasia or Hypoplasia:

- It may be total or partial.
- It might cause cretinism (if total).

2. Ectopic Thyroid:

⇒ Definition:

- Presence of thyroid tissue in a site other than normal position.
- It may be the only thyroid tissue or with normal thyroid gland.

⇒ Sites:

1. May be present along the course of the thyroglossal tract.

DD: Thyroglossal Cyst

1. Clinically:

- It is solid → differentiated by Paget's test
- Thyroid moves with deglutition; but thyroglossal cyst mobile with deglutition & protrusion of tongue.

2. By investigations:

- U/S → cystic or solid.
- Radioisotope scanning to know if there is another normal thyroid tissue or not.

2. Lingual thyroid:

- It's the commonest site
- C/P → Tongue swelling which may lead to dyspnea, dysphagia and dysarthria
- Tc scan → to confirm whether it's the only thyroid tissue.

It may turn malignant → papillary carcinoma

3. Retro-sternal (Intra-thoracic type):

- C/P → Pressure manifestations (especially if becomes goitre)
- CXR, CT & Tc scans.
- TTT → surgical removal

⇒ Treatment:

- If there is normal thyroid tissue → Remove the ectopic one.
- If not:
 - Leave the ectopic gland.
 - Remove it & re-implant it in the anterior abdominal wall to avoid re-exploration with its complication in cases of recurrence. If re-implantation failed, give hormonal therapy for life.
 - Medical thyroidectomy by L-thyroxine to suppress the TSH, this helps to reduce the size of the ectopic thyroid tissue.

3. Lateral Aberrant Thyroid:

- It is a wrong name it is L.N. metastasis of papillary carcinoma and not a congenital anomaly.



4. Thyroglossal Cyst:

⇒ Etiology:

- From unobliterated portion of thyroglossal duct (which develop from foramen caecum and descend into the neck to give thyroid isthmus and medial parts of the lobes.
 - At any point of the course of thyroglossal tract.
 - **The commonest site is "subhyoid in midline"** then on (thyroid cartilage usually pushed to left side as thyroglossal tract is pushed to one side at thyroid cartilage) and rarely "suprahyoid"

- **Etiology:** failure of obliteration
- **Site:** subhyoid>thyroid cart.>suprahyoid
- **C/P:** mass, pain if complicated
- **Comp.:** as cyst + fistula
- **Invest.:** U/S
- **TTT:** Sistrunk operation

⇒ Pathology:

- Wall of cyst: may contain thyroid tissue.
- Lined with: stratified squamous epithelium.
- Containing: sebaceous material.
- Fibrous cord: connect the cyst to hyoid bone (it can be followed to foramen caecum at the base of the tongue).



⇒ Clinical Picture:

- Type of patient: child 6-8 years presented by swelling in the front of the neck.
- Symptoms:
 - 1- Painless mass in the midline of the neck.
 - 2- Pain if infected (the commonest complication as **it is rich in lymphatics**)
- Signs:
 - 1- Cystic mass in midline of the neck (by Paget's test).
 - 2- Mobility:
 - Moves with deglutition and protrusion of the tongue (due to its relation to hyoid bone)
 - Moves from side to side not from above downwards.
 - 3- There may be a palpable track extending from hyoid bone upwards towards tongue.

⇒ Complications:

- 1- As any cyst (**infection**, rupture, hemorrhage, pressure, calcification)
- 2- Thyroglossal fistula:

Etiology: Acquired fistula (**never congenital**) due to:

- 1- Infection of thyroglossal cyst leading to rupture.
- 2- **Inadequate** excision of the cyst.

C/P: - Discharges viscous fluid or pus.
 - The opening is near midline or to the Lt. side **crescentic** due to fibrosis.

⇒ DD: ectopic thyroid.

⇒ Investigations: U/S → cyst.

⇒ Treatment: **Sistrunk** operation:

- Excision of:
 - Cyst.
 - Track.
 - Central part of hyoid bone (to avoid recurrence).
 - Core of tissue of the tongue up to foramen caecum.
 - Pyramidal lobe of the thyroid.

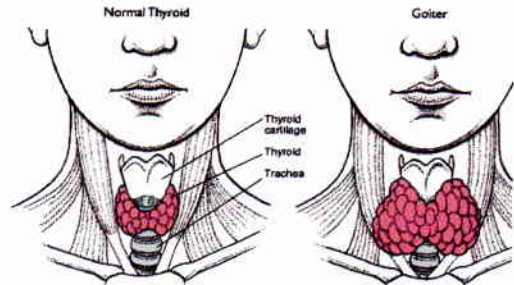
⇒ Prognosis: If incomplete excision → recurrence or fistula may result.

Goitre

"Latin - Gutter = the throat"

Definition

- It is generalized enlargement of thyroid gland.
- NB.** A discrete swelling in otherwise normal thyroid gland is termed Solitary "isolated" swelling.



Classification

1. Simple Goitre

- a. Simple physiological goiter.
- b. Simple colloid goiter.
- c. Simple nodular goiter.

2. Toxic Goitre

1. Primary or diffuse toxic goitre.
2. Secondary or Toxic nodular goitre.
3. Toxic nodule.
4. Rare causes (thyrotoxicosis factitia, Jod-basedow,....etc)

3. Inflammatory (Thyroiditis)

1. Acute bacterial thyroiditis (rare)
2. Sub-acute thyroiditis (De Quervain's thyroiditis)
3. Chronic thyroiditis e.g TB, syphilis (rare)
4. Auto- immune thyroiditis
 - Hashimoto's disease.
 - Riedel's thyroiditis.
5. Collagen disease as Riedel's disease.

4. Neoplastic Goitre

1. Benign
2. Malignant.

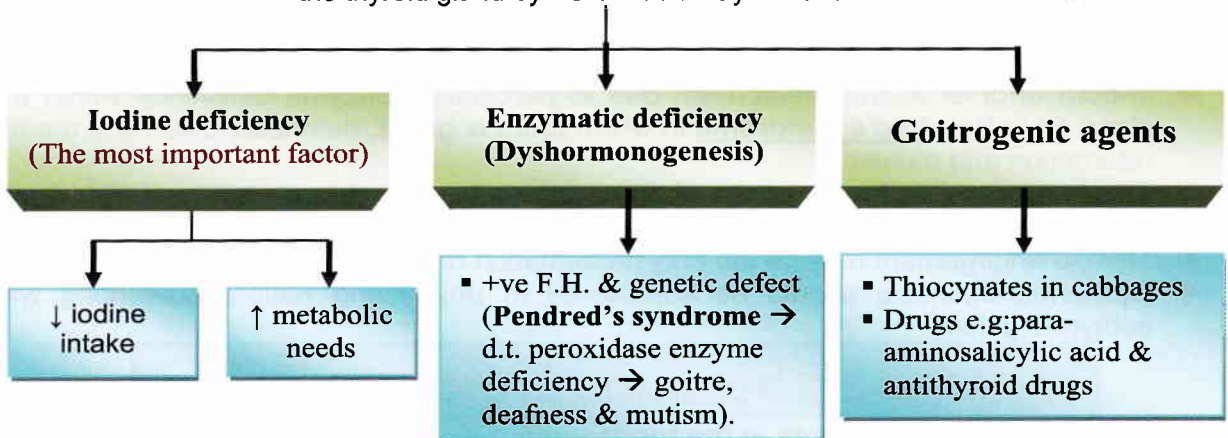
A. Simple Goitre

Definition

- **Non inflammatory, non neoplastic & non toxic enlargement of the thyroid gland (euthyroid).**

Etiology

Simple goitre may develop as a result of stimulation of the thyroid gland by TSH which may increase



1. Endemic Goitre (absolute iodine deficiency)

- **Definition:** when more than 10% of the population of the community is affected in a district.
- **Incidence:**
 - Age: 15-25
 - Sex: female.
 - In Egypt it is seen in the Oases.
- **Etiology:**
 - It is due to **deficient intake of iodine** in the food (**daily need is 0.1-0.15 mg/day**).
 - **Investigations:**
 - We assess the severity of the condition by estimation of iodine-creatinine clearance in urine
- **Treatment:** In endemic areas **iodized table salt** is used (K iodide 1/100000)

2. Physiological Goitre (Venus neck)

- It may occur in **female at puberty or during pregnancy and lactation**.
- It is due to ↑ body demand, which will lead to relative iodine deficiency → relative ↓ of thyroid hormones → ↑ TSH level → stimulation of thyroid gland → diffuse hyperplastic goitre.
 - Patient complains of mild enlargement of the gland
 - **O/E:** gland is diffusely, symmetrically enlarged, Smooth, painless, mobile, with no toxic manifestations.
- **Prevention:** use iodized table salt (potassium iodide 1 : 10000)
- **Treatment:**
 - No treatment (mild cases)
 - Thyroid hormone is given, L- thyroxin 0.2 mg / day for several months to be tapered to 0.1 mg / day for several years.

3. Colloid Goitre

- **Etiology:** is a late stage of diffuse hyperplasia when TSH stimulation has fallen off and when many follicles are inactive and full of colloid as patient may receive large doses of iodine → it will inhibit TSH and protease → hyper-involution of the gland.
- **C/P:** The gland is diffusely enlarged (Soft, Smooth, Symmetrical).
- **Fate:** It may return to normal or cause pressure manifestations.
- **Treatment:** Conservative unless causing pressure manifestations → Subtotal Thyroidectomy.

4. Dyshormonogenesis

- These are usually inherited as autosomal recessive disorders
- In-born error of iodine metabolism due to peroxidase enzyme deficiency within the gland as in **Pendred's syndrome** in which there is **goitre, deafness, dwarfism, mental retardation** and mutism.
- This is responsible for the cases of goitre seen after birth & is usually associated with hypo-thyroidism.
- Thyroid enlargement may be the only presentation of dyshormonogenesis
- Dyshormonogenesis should be considered in any young patient presenting with euthyroid goiter.

5. Sporadic Goitre

- **Due to goitrogenic agents in the food:**
 - **Cabbage.**
 - **Water pollution with excreta.**
 - **Drugs: anti-thyroid drugs, anti TB, calcium.**
- **PASA.**
- **Vegetables of brassica family.**
- **Thiocynate & perchlorates.**

6. Simple Nodular Goitre (most common thyroid disease)

Incidence

- The commonest disease of thyroid gland
- **Age:** 30 - 40 years
- **Sex:** female > male (because of the presence of estrogen receptors in the thyroid gland)

Etiology

- Repeated fluctuations of TSH level (due to repeated cycles of stress) producing mixed patterns within the glands, with areas of active follicles and others with inactive follicles
- As a result of recurrent hyperplasia and hypervascularity, hemorrhage may occur producing necrotic nodules
- Repetition of hyperplasia and involution results in nodular Goitre, most nodules are inactive and the active follicles are present in inter-nodular tissue.

Pathology

- Multiple nodules.
- Sometimes one of the nodules is prominent, while rest of nodules are not palpable (diagnosed as having a solitary thyroid nodule).

- **Etiology:** fluctuations of TSH level
- **C/P:** cosmetic, pressure, pain
- **Comp.:** as cyst + 2ry thyrotoxicosis + retrosternal extension
- **Invest.:** diag., comp., pre-op.
- **TTT:** partial, subtotal or total thyroidectomy

Clinical Picture

Type of patient

- Female 30-40 years with nodular swelling on the front of the neck.

Symptoms

1. **Cosmetic deformity (main complaint)** (painless slowly growing neck swelling).

2. **Pressure symptoms:**

- a) Pressure on trachea → positional dyspnea and cough especially at night
- b) Pressure on esophagus → dysphagia.
- c) Pressure on carotid artery → dizziness.
- d) Pressure on internal jugular vein → blackout increases on leaning forward.
- e) Pressure on RLN → change of voice.

Retrosternal extension or malignancy

Horner's syndrome occurs only in malignancy due to pressure on / or infiltration of sympathetic chain.

3. **Pain:**

- It is uncommon but may be felt when **hemorrhage** occurs in a nodule or late **malignancy**.

Signs

General: no thyrotoxic manifestations.

Local:

⇒ **Clinical Picture of the disease:**

1. **Inspection:**

- Asymmetrical thyroid swelling in lower part of the front of the neck moving up & down with deglutition

2. **Palpation:**

- **Thyroid swelling:**
 - 1- Nodular (solitary, or multinodular).
 - 2- Firm.
- The trachea may be displaced from the middle line or tracheomalacia detected by **Kocher's test**.
- Carotid artery: pulsation is felt but may be shifted in huge goitre.

⇒ **If it is associated with retrosternal goitre there will be:**

1. By inspection: engorgement of the neck veins and dilated veins on chest (due to SVC obstruction).
2. By palpation: lower edge can't be reached.
3. By percussion: dullness over the manubrium sterni.

Complications

1. **Pressure on trachea:**

- Unilateral compression → kinking of trachea.
- Bilateral compression → antero-posterior slit (scabbard trachea).
- Long standing nodular goitre → absorption of tracheal rings (tracheomalacia or chondromalacia), collapse.

- Absorption of cartilage due to long standing pr over trachea by thyroid swelling → dyspnea
- Can be detected by Kocher's test.

2. **Secondary thyrotoxic changes:**

- It occurs in about 30% of cases.



3. Hemorrhage:

- Precipitated by cough or shout
- Considered as emergency case due to sudden compression on trachea → reflex spasm of pretracheal muscles → acute respiratory obstruction and impending suffocation.
- Treatment: urgent aspiration or even emergency subtotal thyroidectomy.

4. Cyst formation: very common.**5. Infection:** very rare.**6. Retrosternal extension.** → which is an indication of operation**7. Calcification (in long-standing cases).****8. Malignant changes** → follicular carcinoma (3%).

Dominant or rapidly growing nodules in long standing goiters should always be subjected to aspiration cytology.

Investigations**1. For Diagnosis:****1. Thyroid function tests:**

- Differentiated from mild toxic nodular goitre.
- Normal T3 & T4 levels
- The presence of thyroid antibodies to differentiate from autoimmune thyroiditis.
- In secondary thyrotoxicosis: ↑ T3, T4 and ↓ TSH

2. U/S of the neck:

- To detect cystic or solid.
- To detect solitary from multinodular.

3. FNABC: only required for a dominant swelling in generalized goitre to exclude malignancy.**2. To exclude complications:**

1. Thyroid scan: to exclude secondary thyrotoxicosis and retrosternal extension.
2. Plain X-ray: to exclude scabbard trachea, calcification and retrosternal extension.

3. Preoperative investigations:

- CBC, KFTS, LFTs, FBS.
- Indirect laryngoscopy for medicolegal importance.

Treatment

■ Surgery is not advised below 25 years (Kasr El-Einy book)

1. Treatment of Simple Nodular Goitre:

Most patients with multinodular goiter are asymptomatic and do not need operation.

Aim of the operation:

1. To **relieve the pressure** symptoms if present.
2. To **prevent complications**.
3. For **cosmetic** purposes.

Most authors agree upon the irreversibility of multinodular goitre, thus surgical excision is the best treatment. However, others agree that half of benign nodules may regress in a period of 10 years spontaneously

1. Procedures:

a- **Partial thyroidectomy:**

- Removal of the nodular parts leaving an equivalent of 8 gm of relatively normal thyroid tissue (size of normal lobe) on each side if feasible to reduce the risk of hyperparathyroidism that accompanies total thyroidectomy.
- We preserve posteromedial part to avoid injury of RLN and parathyroid glands & to preserve part of the gland for secretion of thyroxine.

b- **Subtotal thyroidectomy:** Removal of thyroid tissue leaving about 4-5 gm of thyroid tissue on each side, so, total remnant on both sides equal one normal lobe

if one lobe is more significantly involved than the other, total lobectomy on the more affected side with either subtotal resection or no intervention on the less affected side (**Dunhill procedure**)

c- **Total thyroidectomy:**

- (Total thyroidectomy + replacement therapy) to prevent recurrence and to avoid accidental malignancy. for this reason surgeons favor total thyroidectomy in younger patient as reoperation is hazardous.

d- **Other lines of treatment:**

- In a small-sized swelling (in age < 25 years old) surgery not advised as it may be followed by recurrence (conservative treatment: suppressive dose of L-thyroxine)
- Recently: alcohol injection in the nodules, as it causes necrosis of the nodules and reduction of the size of the gland.

2. Post-operative:

- Suppressive dose of L-thyroxine (0.1 - 0.2 mg/day) to avoid recurrence (prevent TSH fluctuation) in the left thyroid tissue

2. Treatment of Complications:1. **2ry toxic goitre:**

- If < 45 years → Subtotal thyroidectomy after preparation.
- If > 45 years → Radio-active iodine.

2. **Malignant changes** (Follicular carcinoma):

- Total or near total thyroidectomy.
- Supplementary L-thyroxine.
- Radioactive iodine for metastasis.

3. **Retrosternal extension:** surgical excision.4. **Hemorrhage:** urgent aspiration or even emergency subtotal thyroidectomy.

More details

↳ **Histopathological surprise:**

❖ **Definition:**

- Accidentally discovered malignancy from histopathological report of a removed benign thyroid lump

❖ **Treatment**

- If follicular carcinoma: completion thyroidectomy (re-operation to remove the remaining thyroid tissues to facilitate radioactive iodine scanning and destruction of any metastatic foci)
- If papillary carcinoma → re-operation with total thyroidectomy as the tumor is multicentric and excision of juxta-thyroid LN

B. Thyrotoxicosis

Definition

- It is a clinico- pathological condition due to excess thyroxine.
- This ↑ **sensitivity of the tissue to circulating adrenaline.**
- ↑ **Number of Beta receptors.**

- **Hyperthyroidism** = increased thyroxine due to thyroid gland hyperactivity.
- **Thyrotoxicosis** = increased thyroxine due to thyroid gland or any other cause e.g. ectopic hormone production or drug induced.

Causes

A. Common causes

- 1- Diffuse toxic goitre (Primary thyrotoxicosis OR Graves' disease) 76%
- 2- Toxic nodular goitre (Secondary thyrotoxicosis OR Plummer's disease) 14%
- 3- Toxic adenoma (Autonomous nodule) 5%

B. rare causes

1. Neonatal thyrotoxicosis:

- May occur in newborn of thyrotoxic mother due to transmission of thyroid antibodies across the placenta, the condition subsides in 3-4 weeks (decline of the titre of the antibodies in the babies serum)

2. Inflammatory:

- **Hashi-toxicosis:** in early stage of Hashimoto thyroiditis, about 5% of patients have thyrotoxicosis.
- **De Quervain thyroiditis:** due to release of hormones from destroyed thyroid tissue

3. Drug-induced:

- **Thyrotoxicosis factitia:** due to excess intake of L-thyroxine (> 0.3 mg/day)
- **Jod-Basedow thyrotoxicosis:** due to intake of large doses of iodine in treatment of hyperplastic endemic goitre (it is a temporary condition)

4. Tumors:

- Functioning secondary carcinoma
- TSH secreting adenoma of pituitary.
- Struma ovarii: cancer ovary secretes TSH
- Vesicular mole.

- Thyrotoxicosis has been described with a normal T4 concentration (T3 thyrotoxicosis).
- T4 pseudothyrotoxicosis is occasionally seen in critically ill patient and is characterized by increased level of T4 and decreased level of T3 due to failure to convert T4 to T3.

I- Primary Thyrotoxicosis (Graves' disease)

(76 % of thyrotoxicosis)

Definition

- It is an autoimmune disease (type I hyperthyroidism) caused by thyroid-stimulating antibodies (IgG).

Pathology

- Autoimmune disease (Type V hyperthyroidism)
- Occurs on top of normal gland.
- Follicular cells & thyro-globulin (sequestered antigen) are surrounded by membrane preventing its exposure to immune system.
- On stress (psychic trauma or infection) → exposure to immune system with formation of auto-antibodies (Thyroid stimulating antibodies) against TSH receptors & have TSH like action → release of cAMP which ↑ production of thyroxine (↑ all steps of synthesis)

- **Etiology:** autoimmune
- **C/P:** toxic manif. in young ♀ (20-30y)
- **Comp.:** HF, crisis
- **Invest.:** diag., pre-op.
- **TTT:** **MEDICAL**, surgical, radio I & special problems

Macroscopic Picture

- Diffuse enlargement of the whole thyroid gland.

Microscopic Picture

- 1- There is proliferation of the epithelial lining of the acini which may be arranged in several layers.
- 2- The cells are columnar and full of granules.
- 3- The acini contain less vacuolated colloid or are devoid of it.
- 4- There is marked increase in vascularity making surgery if indicated difficult.
- 5- Extensive lymphocytic infiltration is present.

Thyroid stimulating antibodies were previously called long acting thyroid stimulator (LATs)

Clinical Picture

The disease has abrupt onset and shows remission and exacerbation

Type of patient

- Commonly in **young female** (♀ : ♂ = 8 : 1) subjected to psychic trauma, acute infection, pregnancy or labor.
- 50% of patients give family history of autoimmune endocrine disorders

Most Frequent Manifestations

⇒ Symptoms:

- Fatigue, emotional lability, heat intolerance, weight loss, excessive appetite and palpitation

⇒ Signs:

- Tachycardia, hot moist palms, exophthalmos, lid lag and agitation.

For descriptive purpose clinical picture can be classified into

⇒ Symptoms:

- CNS:** irritability, nervousness, insomnia (early symptom), night mares, tremors, **Achillis reflex time is shortened.**
- CVS:** palpitation, dyspnea (if heart failure).
- Metabolic** (most important symptom to differentiate it from psychoneurosis).
 - Increase appetite with loss of weight (**thyroid paradox**).
 - Heat intolerance.

CNS manifestations are more common due to younger age.

- **Eye:** protrusion of the eye ball, diplopia due to weakness of extraocular muscle
- **Skin:** ↑ sweating.
- **Muscle:** easy fatigability (Thyrotoxic Myopathy).
- **GIT:** diarrhea.
- **Sexual :**
 - **Female:** menorrhagia, late amenorrhea.
 - **Male:** impotence.
- **Urinary:** polyuria.
- **Rarely:** painless neck swelling.
- **Other autoimmune diseases** e.g.: type 1 D.M.

⇒ Signs :

❖ General:

- **Mental state:** irritable.
- **BMI:** ↓.
- **Decubitus:** orthopnea if heart failure.
- **Vital data:**
 - Pulse (very important):
 - Rate: ↑.
 - Rhythm: may be irregular (AF).
 - Volume: ↑.
 - Special characters: water hammer pulse (↑COP, ↑systolic pressure, ↓ diastolic pressure → ↓ PR)

Stages of development of thyrotoxic arrhythmias in that order:

- (multiple extrasystoles, then paroxysmal atrial tachycardia, then paroxysmal AF, then persistent AF unresponsive to digoxin)

- Blood pressure:
 - High systolic , low diastolic, high pulse pressure
- Temperature: ↑ in thyrotoxic crisis
- **Eye:** (complications may begin before there is any evidence of thyroid dysfunction or after the hyperthyroidism has been appropriately treated usually, however the ocular manifestations develop concomitantly with the hyperthyroidism)
 - True exophthalmos.
 - **+ ve eye signs:**
 - **Dalrymple's sign** → appearance of rim of sclera above the cornea.
 - **Stellwag's sign** → staring look with infrequent blinking (lid retraction).
 - **Von Graefe's sign** → lid lag on looking down without moving the head.
 - **Mobius's sign** → failure of convergence or to maintain convergence on looking at a near object.
 - **Joffroy's sign** → lack of forehead wrinkling on looking up without moving the head.
 - Others → **Rosenbach's sign** (Tremors on closure of eye).
→ lagophthalmos.

Eye signs may be unilateral



Von Graefe's sign

- **Upper limb:**

- Nail changes :
 1. Brittle.
 2. Spooning
 3. Pitting.
 4. Fissuring.
 5. Thin.
 6. Exaggerated capillary pulsations.
 7. Onycholysis → Separation of nail from its bed.
 8. Plummer's nail → distal end of nail is covered by skin.
- Acropachy.
- Fine tremors.
- Palmer erythema.
- Moist warm sweaty skin.



- **Lower limb:**

- Pretibial myxedema (non pitting edema)
- Pitting edema if heart failure
- Myopathy of proximal muscles

- **Cardiac examination:**

- Accentuated heart sounds
- S3

- **Abdomen**

- Hepatosplenomegaly



❖ **Local:**

- **Inspection:**

- symmetrical swelling in the front of the neck
- Moves upward with deglutition.
- May show expansile pulsation.

- **Palpation:**

- Swelling :
 - Symmetrical.
 - Diffuse.
 - Non tender.
 - Freely mobile.
 - Firm.
 - Thrill may be present.
 - Overlying skin is warm.

- **Auscultation:**

- Bruit over upper lateral part of the gland (over superior thyroid artery)

Complications

- High cardiac output failure.

- **Thyrotoxic crisis.**

⇒ **Definition:** Severe postoperative hyperthyroidism due to inadequate preoperative preparation

⇒ **Clinical picture:** Hyperpyrexia, Hypertension, delirium & convulsions.

⇒ **Treatment:** Cooling + IV (Indral, Corticosteroid, Propylthiouracil, Antipyretics)

DD

1. From other causes of hyperadrenalism: pheochromocytoma, anxiety.
2. Between Grave's and Plummer's.

Investigations

1. For Diagnosis: (most cases are diagnosed clinically)

1. Thyroid function tests:
 - Free T3 & T4 → high.
 - TSH → suppressed.
2. U/S of the neck: mild diffuse enlargement.
3. Thyroid scan: diffuse ↑ uptake and exclude retrosternal goitre.
4. Thyroid Antibodies: TSI.

T3 thyrotoxicosis should be suspected if the clinical picture is suggestive with normal T4 and suppressed TSH

2. Preoperative Investigations:

1. CBC, LFTs, KFTs, FBS, CXR and ECG.
2. Indirect laryngoscopy. (for medicolegal purpose)

To exclude thyroiditis:

- A- Hashimoto → anti-microsomal & anti-thyro-globulin antibodies.
 B- De Quervain → good response to small dose of prednisolone (therapeutic test).

Treatment

I-Treatment of Graves' Disease

A. Medical Treatment: (main line of treatment):

a) Thiourea Group:

	Neomercazole	PropylThiouracil
➡ <u>Action</u>	Inhibits peroxidase & iodine binding to tyrosine.	
➡ <u>Dose</u>	1- 10 mg (2 tablets) 3 times daily, (maximum dose is 60 mg / day) When the patient reaches euthyroid state, the dose is gradually reduced to 5 mg 2 or 3 times daily for 12 – 18 months.	Tablet (50 mg) 2 x 3 (two tablets / 8 hrs) N.B. It is used during pregnancy as it crosses placenta to less extent.
➡ <u>Side Effects</u>	The most dangerous complications are <u>agranulocytosis</u> and <u>hepatotoxicity</u>	

b) B-blockers (propranolol): (it protects the heart)

- Used with neomercazole acts after 2 weeks till the patient becomes euthyroid → stop B-blocker
- Dose: 80-160 mg /d.

c) Diazepam: 5-15 mg /d. if severe CNS manifestations.

B. Surgical Treatment:

- ⇒ Subtotal thyroidectomy after preparation
- ⇒ Indicated in: failure of medical treatment, RSG or huge-sized goitre.
- ⇒ N.B. Some authors believe that total thyroidectomy is better than subtotal thyroidectomy to avoid progression of exophthalmos.

C. Radioactive Iodine:

- ⇒ Using I^{131} (10 millicuri), I^{132} , I^{128} or I^{123} .
- ⇒ **Indicated in:** old patient > 45 years with failure of medical treatment.
- ⇒ **Commonest complication:** myxedema.

	Medical treatment	Surgical treatment	Radioiodine
Advantages	No Surgery & no use of radioactive materials	The cure is rapid & the cure rate is high if surgery has been adequate	No Surgery & no prolonged drug therapy.
Disadvantages	Treatment is prolonged & failure rate is at least 50% (It is impossible to predict which patient is likely to go into a permanent remission)	- Myxedema - 5 % Recurrence - Permanent hypoparathyroidism - Nerve injury - Cosmetic scar	A. Overdose → myxedema - Low dose → recurrence. B. Isotope facilities must be available C. Teratogenicity, D. Carcinogenicity. N.B there is no evidence that therapeutic radio iodine is teratogenic or carcinogenic

I. Treatment of Special Problems

A. Obstetric problems :

1-Toxic goitre in pregnancy:

- a. Radio- active iodine is contraindicated, because it is teratogenic and will destroy fetal thyroid.
- b. Anti- thyroid drugs should be used in minimum doses supported by B-blockers
 - In 3rd trimester: antithyroid drugs + small dose of L-thyroxine to suppress maternal TRF o avoid transmitted thiouracil goitre.

- c. Surgical treatment (if indicated): it is better to be performed in 2nd or 3rd trimester.
- d. During Lactation: propylthiouracil is recommended.

2. Postpartum hyperthyroidism:

Pregnancy may lead to exacerbation of a variety of autoimmune disorders in the post-partum period

B. Toxic goitre in children:

- a. Anti- thyroid drugs waiting for spontaneous remission (the ideal).
- b. Surgery (near total) is not preferred as it may be followed by either:
 - High recurrence rate due to rapid growth rate.
 - Hypothyroid state which affects later growth.
- c. Radio- iodine is absolutely contraindicated because of the risk of producing carcinoma of thyroid.

C. True exophthalmos:

1. Treatment of the primary thyrotoxicosis:

Medical:

- Antithyroid drugs + small doses of L-thyroxine to suppress TSH and EPF.

Surgical:

- If subtotal thyroidectomy is indicated, exophthalmos should be stationary for 6 months.

2. Treatment of the exophthalmos:

Medical

- Head up during sleep to decrease eye congestion.
- Protection of the eye by day → eye drops & by night → ointment.
- Dehydrating measures (diuretics).
- High doses of prednisone (local administration is risky especially in presence of venous congestion).

Surgical

- Lateral tarsorrhaphy (it does not prevent the progression).
- Orbital deroofing (Naffziger operation).

D. Thyrocardiac patient:

- The cardiac condition takes priority in management.
- Thyroidectomy is ideal after control of the cardiac status.
- If it is not permissible, radioiodine is used followed by antithyroid drugs until the effect of the former appears (6 weeks)

Secondary Thyrotoxicosis (Plummer's Disease)

(14 % of thyrotoxicosis)

Definition

- Excess secretion of thyroid hormones from active internodular tissue of simple nodular goitre.

Pathology

- Occurs on top of long standing simple nodular goitre.
- The nodules are inactive & internodular tissues are active, however occasionally one or more nodules become active

- **Etiology:** longstanding SNG
- **C/P:** toxic manif. In 40 y. ♀ & no immune manif.
- **Invest.:** diag., pre-op.
- **TTT:** SURGICAL (after prep.), radio I & special problems

Clinical Picture

The disease has gradual onset and slowly progressive course

Type of Patient

- Commonly in females > 40 years with past history of thyroid swelling of long period followed by toxic manifestation.

Most Frequent Manifestations

As Graves'.

Cardiac manifestations are more common due to older age group

For descriptive purpose clinical picture can be classified into

A- Toxic Manifestations

- As in Graves' disease but cardiovascular manifestations are more prominent due to age.

B- Local manifestations (Thyroid Gland)

1. The gland is large & **nodular**.
2. **Firm** in consistency.
3. **No pulsation and no thrill**.

There are no autoimmune manifestations.

DD

As Graves'.

	Graves' disease	Toxic nodular goitre
Age	Young	Elderly
Onset	Abrupt	Gradual
Course	Exacerbations & remissions	Steady
Nervous symptoms	+++	
Metabolic manifestations	+++	+
Cardiovascular manifestations	+	+++
Eye signs	Exophthalmos	Usually no exophthalmos
Thyroid	Diffuse enlargement soft & vascular	Multiple or solitary nodules

Investigations

For Diagnosis

1. Thyroid function: as Graves'
2. U/S of the neck: multiple nodules.
3. Thyroid scan: nodules are cold and internodular tissue is overactive.

Preoperative Investigations as Graves'.

Treatment

I- Treatment of Secondary Thyrotoxicosis

1. Surgical Treatment (main line of treatment)

Subtotal thyroidectomy after preparation as the gland is very large.

2. Radio-active Iodine:

- Less effective as fibrosis will prevent penetration but it can be used only in high risk patients e.g. heart failure.

NB: Medical Treatment: Has no effect as the disease has progressive course so, it is used only as a preoperative preparation.

II- Treatment of Special Problems

1. Toxic goitre with severe cardiac damage (Thyro-cardia)

- a. Control the cardiac problem.
- b. Thyroidectomy is ideal.
- c. In high risk conditions, radio-active iodine followed by anti-thyroid drugs

2. T3 Thyrotoxicosis: TTT → as thyro-cardia.

Toxic Nodule (Autonomous Nodule)

Definition

- Excess secretion of thyroid hormone from autonomous hyperactive thyroid nodule

Pathology

- Solitary hyperactive autonomous nodule (not due to thyroid antibodies)
- The nodule suppresses TSH secretion leading to suppression of rest of the thyroid gland which becomes atrophic.

Clinical Picture

The disease has gradual onset and slowly progressive course.

Type of Patient Commonly in females at any age

Most Frequent Manifestations

- As Graves' but CVS or nervous manifestations according to age.

For Descriptive Purpose Clinical Picture Can be Classified into

A- Toxic Manifestations as Graves'.

B- Local Manifestations (Thyroid Gland)

- Only one palpable nodule is felt in the gland.

DD as Graves'.

There are no autoimmune manifestations.

Investigations

For Diagnosis

- Thyroid Functions: as Graves'.
- U/S of the neck: solitary nodule
- Thyroid scan: hot nodule with suppression of the uptake of surrounding thyroid tissue.

Preoperative Investigations as Graves'.

Treatment

1. **Surgical Treatment:**

- Ipsilateral total lobectomy.
- Indicated in patients < 45 years.

2. **Radioactive iodine.**

- Very effective (as the autonomous nodule is the only part that will take the iodine). But used in patient > 45 years for fear of malignancy

3. **Medical Treatment:** as secondary thyrotoxicosis.

Diagnosis of Thyrotoxicosis

➤ **Clinical examination provides adequate basics for toxicity, should be suspected in :**

- Children with growth spurt.
- Elderly persons with unexplained tachycardia.
- Unexplained diarrhea.
- Unexplained loss of weight.
- Refractory heart failure.

More Details of Clinical Picture

Q. why diarrhea?

Due to increased c-AMP which increases permeability of cells of the mucous membrane.

Q. why polyuria?

due to:

1. ↑ COP which causes ↑ renal blood flow → ↑ GFR.
2. Secondary DM → Glucosuria.
3. ↑ Metabolic water.
4. ↑ Water intake secondary to polyphagia.

Q. why menorrhagia, Then amenorrhea?

Menorrhagia (due to disturbance of estrogen metabolism → abnormal shedding of the endometrium → menorrhagia) Then amenorrhea (due to drop of estrogen level)

Q. why water hammer pulse?

Due to ↑ Systolic due to ↑ CO & ↓ diastolic (accumulation of metabolites → vasodilatation → ↓ peripheral resistance)

Thyroid acropachy:

Acral changes associated with thyrotoxicosis:

- Tremors.
- Pretibial myxedema. (due to deposition of hyaluronic acid in the dermis and subcutis usually in the areas of trauma ccc by thickening of the skin)
- Nail changes.

Effects of thyrotoxicosis on bone:

- Severe thyrotoxicosis causes osteoporosis
- After operation, the osteoporotic bone will withdraw the Ca from blood; this is called **BONE HUNGER** & is manifested by tetany in the post-operative period.

Thyrotoxicosis should always be considered in :

- Children with a growth spurt, Behavioral problems or myopathy.
- Tachycardia or arrhythmia in the elderly.
- Unexplained diarrhea.
- Loss of weight.

More Details of Treatment

1. Medical Treatment

B. Aim:

- Reach the euthyroid state.
- Permanent remission by maintenance dose for prolonged period

C. Indications:

1. 1st try thyrotoxicosis.
2. Mild or moderate toxicity
3. Children & young patients
4. Preoperative preparation (in 2nd cases & toxic nodules < 45 y.)
5. Postoperative recurrence.
6. Contraindications for surgery.

D. Advantages:

- Avoid surgical risks
- Avoid radiation

E. Disadvantages:

- We can not predict if the patient is liable to pass into remission or not
- High recurrence rate (60% within 2 years from stopping treatment)
- Side effects of the drugs.

F. Types:

- Thiourea Group:

Neomercazole (carbimazole):

The most used drug (as less bone marrow depression)

⇒ Action:

- Inhibits peroxidase → ↓ oxidation of iodide to iodine
- Blocks iodine binding to tyrosine (↓ **coupling**)
- Immune suppressant → ↓ Antibody titre

⇒ Dose:

- 10 mg (2 tablets) 3 times daily, (maximum dose is 60 mg / day)
- When the patient reaches euthyroid state, the dose is gradually reduced to 5 mg 2 or 3 times daily for 12 – 18 months.

⇒ Contraindication:

1. Drug sensitivity.
2. Lactation.
3. Retrosternal goitre

Propylthiouracil

⇒ Used in: pregnancy as it crosses the placenta to less extent

⇒ Action

- Blocks peroxidase enzyme
- Blocks iodine binding to tyrosine
- Prevents peripheral conversion of T₄ to T₃

⇒ Dose:

Tablet (50 mg) 2 x 3 (two tablets / 8 hrs)

➤ It is given until spontaneous cure occurs i.e. drop of TSI.

⇒ **Complications:**1. **Thiouracil goitre** due to increase TSH (increase vascularity)2. **Transmitted thiouracil goitre**

- If given during the last trimester of pregnancy → baby will be born with goitre → face presentation.
- Mechanism: if anti-thyroid drugs are given in late pregnancy → (TRF) of the mother will increase & crosses the placenta to the fetus → thyroid of the baby will be enlarged → extension of the neck.

3. **Compression** on trachea with retrosternal goitre.4. **Skin rash.**5. **GIT upset.**6. **Hematuria.**7. **Arthralgia.**8. **Jaundice** (Hepatotoxic).9. **Agranulocytosis:**

- Manifested by pyrexia & sore throat (1st present.).

- Treatment

- Prophylaxis

- Notify pt. about the above symptoms.

- Blood picture should be performed every 2 wks

- Treatment

- i) Stop the drug.
 - ii) Fresh blood transfusion.
 - iii) Penicillin.
 - iv) Vit. B12 and folic acid.
 - v) Cortisone in severe cases.

- **Propranolol (B blocker)**⇒ **Action:**

- It is blocker for the B-receptors → protect the heart & lower the heart rate (cardioprotective drug).
- Decrease the peripheral conversion of T4 to T3.

⇒ **Uses:**

- In medical TTT of primary toxic goitre with neomercazole till the patient becomes euthyroid → stop Beta-blockers.
- In preoperative preparation either:
 - a-With neomercazole till patient becomes euthyroid.
 - b-Without neomercazole (rapid preparation) esp. in RSG, it should be continued for 1-2 weeks after surgery to avoid post-operative thyrotoxic crisis.

Other Lines of Medical Treatment

Lugol's iodine:

- Mechanism :
 - Reduces the effect of TSH on the thyroid gland.
 - Not used in long-term control and used mainly for preoperative preparation.

K Percolate:

- Mechanism: ↓ iodine trapping.
- Complication: Aplastic anemia.
- Only used if there is allergy to thiourea.
- Dose: 100 -200 mg /6 hours

2. Surgical Treatment

(Subtotal thyroidectomy after preparation)

A. Indications:

- 2ry thyrotoxicosis.
- 1ry thyrotoxicosis with :
 - Failure of medical treatment (No remission after medical TTT).
 - Severe condition from the start (pulse >120/min, as this patient will not go into spontaneous remission).
- Pressure manifestations.
- Retro-sternal goitre.
- Failure or intolerance of medical treatment.

B. Aim:

- Removal of thyroid tissue leaving about 4-5 gm of thyroid tissue on each side, so, total remnant on both sides equal one normal lobe.
- Solitary toxic nodule is treated by *ipsilateral* total lobectomy

C. Preparation:

- Preoperative investigations are required including thyroid function tests, laryngoscopy and it is to be noted that isotope scan is required in toxic nodular goiter if total thyroidectomy is not planned and it is of no value in diffuse toxic goiter because uptake is uniform.
- Carbimazole 10 mg tds + propranolol 40 mg tds till euthyroid state is reached
- Stop propranolol and continue with:
 - Carbimazole 5 mg tds to the evening prior to surgery.
 - Lugol's iodine 15 oral drops tds, for 14 days to make the gland less vascular.
- Block & replace regime may be used instead. (see above)

Propranolol should be continued post-operatively for few days.

Lugol's Iodine

- **Composition:** 5 % iodine + 10 % k iodide in water.
- **Mechanism of action:**
 - It inhibits TSH and protease enzyme so the colloid accumulates in the follicles, which become distended and compress the surrounding blood vessels.
 - So the gland becomes firmer and less vascular.
 - It reaches the maximum effect after 10-14 days then decline, so it should be used during the last 10-14 days prior to surgery.
 - It is used once for life.

How to know that the patient is ready for surgery?

1. Symptomatic relief.
2. Gaining weight.
3. Sleeping pulse. (pulse alone is not an indicator of euthyroid as Inderal will normalize pulse even before action of Neomercazol)
4. Serum T₃, T₄ level.

D. Complications of thyroidectomy:

A. Operative complications:

1. Complications of anesthesia.
2. Shock.
3. Hemorrhage: primary.
4. Injury of important structures:
 1. External laryngeal nerve → loss of high pitched voice (see before)
 2. Recurrent laryngeal nerve → may be partial or complete, unilateral or bilateral.
 3. Trachea.
 4. Esophagus.

B. Post-operative complications:I. Early complications:

1. hemorrhage:

a. Reactionary hemorrhage:

- Due to Slippage of superior thyroid a. ligature.
- The hematoma may compress trachea or carotid vessels.
- Treatment → emergency removal of sutures of skin and deep fascia (in bed) → return to operative room.

b. Secondary hemorrhage.

2. Infection.

3. Pulmonary complications: DVT – pulmonary embolism.

2. late complications:

1. Hypothyroidism: (20 - 40% of cases)

- Rarely due to extensive removal of thyroid tissue but commonly due to alternation of autoimmune process leading to destruction of thyroid tissue.
- Treated by L-thyroxine

2. Recurrent thyrotoxicosis: in less than 5% of cases usually due to inadequate removal.

3. Thyrotoxic crisis (thyroid storm):

- It is life threatening of hyper-acute hyperthyroidism.

- Causes:

1. Spontaneous.
2. Postoperative especially unprepared patients with thyrotoxicosis
3. Rarely, it may be the first presentation of thyrotoxicosis if the patient exposed to stress e.g. infection and surgery.

- C/P: (Sympathetic hyper-stimulation)

- **Hyperpyrexia (> 40°C)**
- **Heart failure**
- Hypertension.
- Coma → death
- Tachycardia (severe)
- Excessive sweating.
- Irritability & convulsions.

- Treatment → emergency:

- **Cooling of patient: ice packs, antipyretics.**
- **Propranolol 1mg IV drip (under monitor)** (may be repeated)
- IV fluids.
- IV hydrocortisone.
- Digoxin for heart failure.
- Sedation.
- Anti-thyroid drugs:
 - o Carbimazole 15-20 mg /6 hours.
 - o Lugol's iodine 10 drops / 8 hours or IV drip of K iodide.

4. Hypo-parathyroidism (in less than 0.5% of cases) → Tetany

- First → Circum-oral numbness
- Then → carpo-pedal spasm & laryngismus stridulus.
- It may be due to: removal or devascularization of parathyroid glands.

5. Keloid scar: if incision overlies the sternum & managed by local steroid injection.

Causes of dyspnea after thyroidectomy:

1. A tension hematoma in deep cervical fascia.
 2. Laryngeal edema due to trauma by endotracheal tube or secondary to extensive manipulation of larynx during surgery.
 3. Bilateral recurrent laryngeal n. injury.
 4. Tracheal copllapse due to tracheomalacia.
- Whenever respiratory obstruction is suspected, an endotracheal tube should be immediately inserted or even a tracheostomy is performed

3. Radio-active iodine

A. Action:

1. I^{131} , I^{132} , I^{128} , & I^{123} when concentrated by the gland will emit **B irradiation**.
2. It destroys the major part of the gland (according to the dose) without affecting the adjacent structures (due to short penetration).
3. It emits **little amount of gamma irradiation**.

B. Indications:

1. Primary thyrotoxicosis in patient above 45 years with failure of medical treatment.
2. High risk patient (as secondary thyrotoxic cases with heart failure).
3. Recurrent thyrotoxicosis instead of the second operation in the neck.
4. Toxic nodules if >45 y. (The autonomous nodule is the only part that will take the radioactive iodine as the rest of the gland will be suppressed due to low TSH).

C. Disadvantages:

1. Overdose → **myxedema** in 75-80% of cases after 10 years.
2. Need isotope facilities.
3. Difficult to calculate the dose as it is according to the weight of the gland.
4. Low dose → Recurrence.
5. Teratogenic → so not given to pregnant women.
6. Carcinogenic so not given below the age of 45 years (it is proved not carcinogenic for 30 years).
7. Less effect in secondary thyrotoxicosis because fibrosis will prevent penetration.
8. **Improvement is expected within 8-12 weeks.**

D. Dose:

- 10 millicurie I^{131} , another dose may be given after 3 months if there is no good response (we use 10 microcuries for isotope scanning).

N.B. Recently, reluctants to prescribe radioactive iodine to those under the age of 45 years has faded in UK

NOTES

Thyrotoxicosis should be suspected in:

1. Children of growth spurt or behavioral problems.
2. Unexplained tachycardia or arrhythmia.
3. Unexplained diarrhea.
4. Unexplained loss of weight.
5. Resistant heart failure.

WAYNE scoring index (obsolete)

3 symptoms

1. Thyroid paradox.
2. Hot intolerance.
3. Palpitation.

11-19 → Doubtful

3 signs

1. CVS.
2. Eye.
3. Swelling and/or metabolic.

> or = 20 → Sure.

Medical Treatment		Surgical Treatment	Radioactive Treatment
Carbimazole 10 mg tds	Propranolol 40 mg tds	Preparation Carbimazole + Propranolol ↓ Euthyroid state ↓ Carbimazole + Lugol's iodine for 2 weeks ↓ Subtotal Thyroidectomy ↓ Postoperative propranolol for few days Rapid Preparation by propranolol for 1 week. ↓ Subtotal Thyroidectomy ↓ Postoperative Propranolol for 2 weeks	
↓			- 10 millicurie of radioactive iodine - 3 months, another dose may be given.
Euthyroid State			
↓			
Carbimazole 5 mg tds for 12-18 months			

Exophthalmos

Definition

Protrusion of the eye ball due to thyroid disease (i.e. thyroid proptosis)

NB: *Proptosis is protrusion of eyeball due to any disease rather than thyroid.*

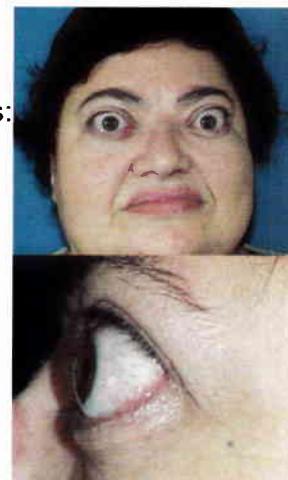
Types

⇒ False (Lid Retraction):

- **Definition:** retraction of Muller's muscle (part of levator palpebrae superioris) as the excess thyroxine increases the sensitivity of Muller's muscle (supplied by sympathetic fibers) to circulatory catecholamines.
- It accompanies both diffuse and nodular goitres.

⇒ True (Actual Proptosis):

- **Definition:** actual protrusion of the eye ball.
- **Etiology:** exophthalmos-producing factor (EPF) which causes:
 - 1- Retro-bulbar edema.
 - 2- Retrobulbar deposition of fat.
 - 3- Weakness of the extra-ocular muscles (external ophthalmoplegia)
- It is characteristic of Graves' disease.
- The condition is aggravated by ophthalmic vein compression → lid edema and conjunctival injection.
- **Malignant exophthalmos:** is a severe progressive form.



Complications

1. Keratitis.
2. Corneal ulceration.
3. Pan-ophthalmitis (if the exophthalmos is rapidly progressive).
4. Retinal damage.

Examination of Exophthalmos

A. To show true or false:

1. **Naffziger's Method:** the examiner stands behind patient with the head tilted backwards to see the level of supra and infra orbital ridge with the cornea.
2. **Frazer's Method:** the examiner stands on one side of patient with the eyes lightly closed and see obliteration of sulcus of orbital margin
3. **Ruler's Method:** see the level of supra & infra orbital margin with cornea by a ruler.

B. To determine the degree:

- 1- Exophthalmometer.
- 2- Ruler: measures distance between lateral orbital margin and apex of the cornea (normally = 15 -17 mm)

Treatment of Exophthalmos

⇒ **Aim of treatment:** → For fear of complication

⇒ **There are 2 parallel lines Should be fulfilled:**

1. Treatment of the primary thyrotoxicosis:

Medical: antithyroid drugs + small doses of L-thyroxine to suppress TSH

Surgical: if subtotal thyroidectomy is indicated, exophthalmos should be stationary for 6 months.

2. Treatment of the exophthalmos

Medical

1. Head up during sleep to decrease eye congestion.
2. Protection of the eye by day → eye drops & by night → ointment.
3. Dehydrating measures (diuretics).
4. High doses of prednisone (local administration is risky especially in presence of venous congestion).

Surgical

1. Lateral tarsorrhaphy (it does not prevent the progression).
2. Orbital deroofing (Naffziger operation).

Exophthalmos is due to EPF which follow TSH i.e. they ↑ together & ↓ together

Retrosternal Goitre

Types

A. Congenital (rare):

1) Plunging goitre:

- The thyroid gland rises with deglutition & then descends again through the thoracic goitre.

2) Intrathoracic goitre:

- The gland completely present in the chest & separated from the main gland.
- It arises from ectopic thyroid tissue which takes its blood supply from mediastinal vessels.

B. Acquired:

1) Mediastinal goitre:

- The nodule completely presents in the chest but connected with thyroid by narrow band of tissue & take its blood supply from thyroid vessels.

Pathology

- Retrosternal goitre usually arises in a normally placed thyroid gland. The sternohyoid and sternothyroid muscles prevent forward expansion, and assisted by gravity and the negative intrathoracic pressure, they direct the swelling into the mediastinum
- It is more common in males due to short neck & strong strap muscles.

Clinical Picture

Retrosternal goitre may be simple, toxic or malignant

Symptoms

- May be **asymptomatic**.
- History of cervical goitre which has disappeared but severe obstructive symptoms present:
 1. Dyspnea, cough & stridor (worse at night & ↑ by neck flexion or lying down).
 2. Dysphagia.
 3. recurrent laryngeal nerve affection is not common
- Toxic manifestations may be found.

Dysphagia with thyroid occurs in:

- 1) Retrosternal goitre.
- 2) Riedel's thyroiditis.
- 3) Malignancy (infiltration of the esophagus).
- 4) Myopathy of striated muscle of the esophagus in toxic patients

Many patients attending the chest clinic with a diagnosis of asthma are proven to be due to retrosternal goiter, this occurs before the true nature of the problem is discovered.

Sings

- **Inspection:** engorgement of neck veins and dilated veins on chest, cyanosis & edema of the face & neck due to compression on superior vena cava .
- **Palpation:** the lower border is not felt.
- **Percussion:** dullness over the manubrium sterni.
- **Special tests:** Pemberton's sign: patient elevates the arm above the level of head, it is considered positive when facial plethora (blue or pink effusion of the neck and/or the face due to venous obstruction).



(A1) At rest, he has moderate puffiness of the face.

(A2) After raising the arms briefly to further obstruct venous drainage, swelling increases and there is marked suffusion of the face.

(B) Woman with an obstructive, predominantly retrosternal goitre, showing dilated anterior chest veins caused by increased collateral venous flow.

Investigations

Imaging:

a. CT scan

The investigation of choice

b. Plain X- ray:

Show soft tissue shadow in the superior mediastinum.

c. Isotope scan:

Will help to differentiate an intra-thoracic goitre from a mediastinal tumor.

Flow volume loop pulmonary function test

The rate of flow is plotted against the volume of air inspired and then expired to detect significant tracheal compression.

DD: of shadow in superior mediastinum:

- 1- Retrosternal goitre.
- 2- Thymoma
- 3- Lymphoma
- 4- Enlarged LN.
- 5- Aortic aneurysm

Treatment

Thyroidectomy is the only line of treatment

- **Subtotal thyroidectomy** from the neck (piecemeal) because the vascularity in the neck but if huge or intrathoracic median sternotomy may be needed.
- If **toxic subtotal thyroidectomy after preparation by Indral** is done but not anti-thyroid drugs to avoid the increase of the size of the gland.

- **Special care should be exerted to avoid injury of RLN during delivery of R.S.G**
- **Fragmentation must be avoided when malignancy is suspected**

C. Thyroiditis

- 1- Autoimmune thyroiditis :
 - a- Goiterous (hashimoto's disease)
 - b- non goiterous (primary myxedema)
- 2- Acute thyroiditis
- 3- Riedel's thyroiditis
- 4- Subacute thyroiditis (de quervain's)
- 5- T.B thyroiditis (occur in system)

Autoimmune Thyroiditis (lymphadenoid Goitre - Hashimoto's Disease)

➔ It is the most common form of thyroiditis.

Etiology

- Due to formation of auto **anti-bodies to thyro-globulin & microsomes**.
- This antigen- antibody reaction → **destruction** of the thyroid follicles.

- **Etiology:** autoimmune
- **C/P:** thyrotoxicosis(5%) → myxedema
- **Comp.:** myxedema, pressure, lymphoma
- **Invest.:** **FNAC**, lab., radio.
- **TTT:** 1.**Medical:**cortisone, L-thyroxine, indral
2.**Surgical**

Antibodies in Hashimoto's thyroiditis destroy the glands, while in primary thyrotoxicosis the antibodies stimulate secretion.

Pathology

Macro

Gland is large & nodular due to granulation tissue

Micro

Acidophilic **Askanazy cells** + heavy lymphocytic infiltration & plasma cells.

Clinical Picture

Type of patient

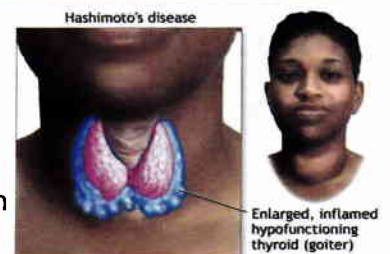
- Middle age female, with goiterous myxedema & other autoimmune disease.

Symptoms

- Fluctuating course.
- Manifestations of thyrotoxicosis in 5% of cases.
- Manifestations of myxedema.
- Other autoimmune disease.

Signs

- **General:**
 - Manifestations of myxedema (20%) & usually associated with splenomegaly.
 - Autoimmune manifestations for example: erythema nodosum .



- **Local:**
 - Inspection :
 - Asymmetrical large swelling in the front of the neck.
 - Moves up with deglutition.
 - Palpation :
 - Large nodular firm asymmetrical swelling.

Differential Diagnosis SNG but, euthyroid

Complications

1. Myxedema.
2. Pressure effects.
3. Lymphoma.
 - Cause: usually non Hodgkin's lymphoma may be Burkitt's lymphoma.
 - 1ry extra-nodal, 2ry if nodal → bad prognosis.
 - TTT: localized to gland → irradiation, with LNs → chemotherapy VAP.

Investigations

- **Laboratory:**
 - Thyroid function ↓
 - Antibody detection : Antithyroglobulin & Antimicrosomal antibody
 - High ESR , leucocytosis
- **Radiological:**
 - U/S: multiple nodules
 - Thyroid scan : low uptake
- **FNABC: Askanazy cells + lymphocytic infiltration**
 (the best investigation although abundant lymphocytes may make the cytological differentiation between autoimmune thyroiditis and lymphoma very difficult)

Treatment

⇒ **Medical Treatment:**

1. Cortisone.
2. L-Thyroxine. (Main treatment)
3. Indral to control toxic symptoms during Hashi-toxicosis.

⇒ **Indications for surgery**

1. Large goitre with pressure manifestations
2. Suspicion of malignancy. i.e :
 - Rapid increase in size.
 - Pain.
 - Ulceration.
 - LN enlargement.

Riedel's Thyroiditis

- Rare 0.05 % of goiters
- It is extensive fibrosis of the neck, thyroid → (**frozen neck** or **woody thyroid**).
- It may be associated retro-peritoneal fibrosis.
- The patient presented by hard nodule in the thyroid & pressure manifestations, so **it simulates malignancy** (DD from anaplastic carcinoma by FNABC).
- Thyroid gland is adherent & sticky to skin.
- May occur on top of Hashimoto or sub acute thyroiditis and in this case:
 - Hypo-thyroidism is usually present.
 - No sclerosing cholangitis.
 - No mediastinal or retro-peritoneal fibrosis.

Treatment

Lahey's operation (Isthmectomy to relief pressure on trachea & to take biopsy)

Acute Thyroiditis

- **Etiology:** bacterial infection, following infection of mouth or pharynx.
- **Clinical picture:** severe neck pain, dysphagia, fever & chills
- **Treatment:** proper antibiotic & drainage of suppuration may be required.

Subacute Thyroiditis (Granulomatous Thyroiditis, de Quervain Thyroiditis)

- Considered to be a viral infection (or as a complication of **Mumps**).
- Microscopy there is giant cells
- There is pain in the knee, liver malaise, firm irregular enlargement of thyroid.
- There is large, red, hot & tender thyroid gland & febrile reaction.
- Thyroid antibodies is absent.
- We might classify the disease into four stages:
 - **Stage I:** Acute toxicity (destroyed follicles) + enlarged tender gland for 2 months.
 - **Stage II:** Euthyroid + enlarged gland, decrease tenderness.
 - **Stage III:** Hypothyroidism + enlarged gland → occur 2 - 4 months from the onset.
 - **Stage IV:** Recovery after 1 - 6 months.

Investigations

- ESR, ↓ iodine uptake of the gland.
- Normal or depressed leucocytic count.

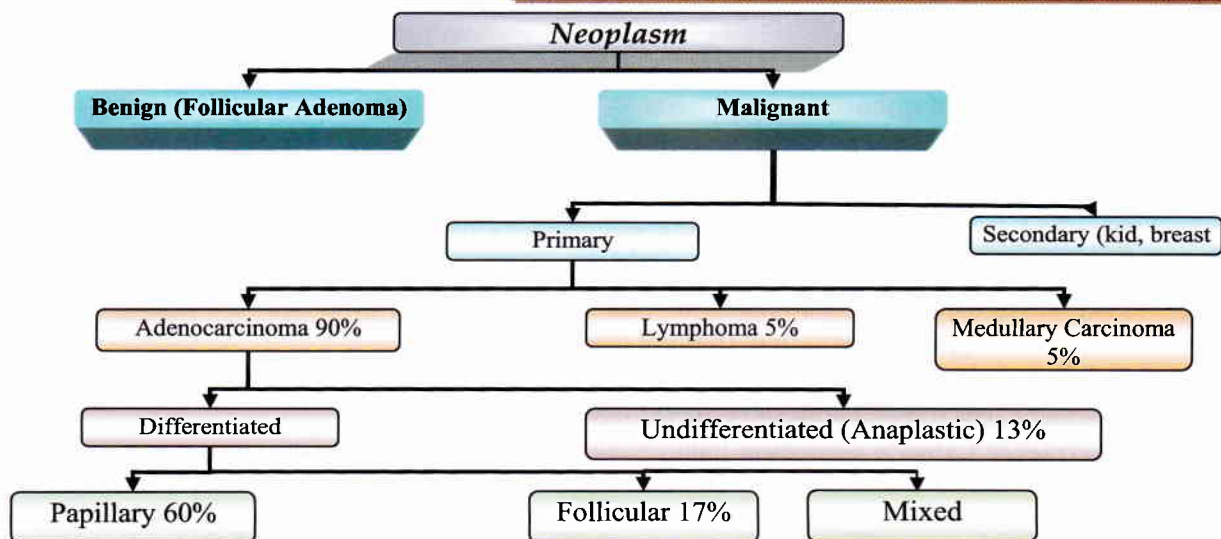
Treatment

- Oral prednisone for few weeks (rapid response is diagnostic).

D. Thyroid Neoplasm

The classification of malignant thyroid tumors into differentiated and undifferentiated is called Dunhel classification

- **Path.:** pap.(60%), follic.(17%), anap.(13%)
- **Comp.:** a. local (infiltration) b. General (metastasis)
- **C/P:** swelling ± pain + infiltration + metastasis
- **Invest.:** diag., staging, pre-op., follow-up
- **TTT:** 1.DTC: radical thyroidectomy + radio I
2.Anaplastic: debulking + radio I
3.Medullary: surgery + ttt of familial type
4.lymphoma: radio + chemo



A. Benign:

1. Follicular adenoma (5 histological subtypes)

1. Embryonal adenoma → without lumen.
2. Fetal adenoma → Small follicles lined with cubical cells containing colloid (little differentiation)
3. Micro-follicular adenoma → Follicles filled with colloid (complete differentiation)
4. Macro-follicular adenoma → Large follicles with much colloid
5. Hurthle cell adenoma: (variant of follicular adenoma)
 - Well formed acini with cells showing granular acidophilic cytoplasm (Askanazy cells)
 - It is liable to turn malignant

Presentation → Solitary thyroid nodule

Investigations

a- U/S → Solitary nodule

b- Thyroid scan → Cold nodule

Treatment → Hemi-thyroidectomy (lobectomy + isthmectomy) & paraffin section (To exclude capsular & vascular invasion)

NB: FNABC cannot detect vascular or capsular invasion.

Papillary adenoma:

- Usually with papillary projection inside (most pathologist consider it as malignant, so there is nothing called papillary adenoma).
- All papillary tumors should be considered as malignant, even if encapsulated.

B. Malignant:

1) Primary:

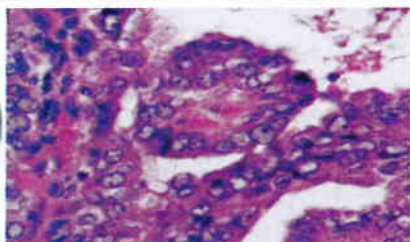
1. Papillary carcinoma.
2. Follicular carcinoma.
3. papillary–follicular carcinoma
4. Anaplastic carcinoma.
5. Medullary carcinoma.
6. Malignant lymphoma.

2) Secondary:

Arise from local infiltration from near by malignancy or blood born.

	Papillary carcinoma	Follicular carcinoma	Anaplastic carcinoma
Incidence	60%	17%	13%
Age.	20 – 40 years (children & young adults)	40 – 60 years (middle aged)	Elderly
Sex (F:M)	3.5 : 1	2 : 1	1 : 1.3
Predisposing Factors	1. External irradiation of neck in children which was previously used for TTT of hemangiomas, T.B lymphadenitis. 2. papillary adenoma 3. genetic factors : a- Godwin's \$ b- ret/PTC3 oncogenes	1. SNG. 2. Follicular adenoma.	Usually De Novo

Multiplicity	Ill-defined mass infiltrating the surroundings		
	- 80% Multicentric due to intrathyroid lymphatic spread	Rare	
Micro	Loss of polarity & signs of mitosis.		
	- Malignant papillae with vascular C.T. core covered by malignant cells - Laminated calcified bodies (psammoma bodies) - The tumors shows characteristic pale empty nuclei (orphan Annie eye nuclei)	- Thyroid follicles with variable degrees of differentiation - Capsular & vascular invasion	- Clusters of spindle cells (small or large) - Separated by little fibrous tissue
Spread	▪ Mainly lymphatic to deep cervical L.N (Aberrant thyroid) ▪ Solid sheets may be present.	Mainly blood to <u>skull</u> usually solitary, painful, pulsating, osteolytic (D.D. abscess)	Mainly direct & can infiltrate carotid artery which may rupture.
TSH	Dependant	Less dependant	Not dependant
Prognosis	Good except if the pt at the peak of 40 years	Bad	Very bad
10 years survival	90%	Encapsulated 97% Invasive 70%	Die within 1-2 years



Orphan Annie eye nuclei

Microcarcinoma (occult carcinoma): the term occult was formerly applied to all papillary carcinoma less than 1.5 cm in diameter, but the preferred terminology now is microcarcinoma for cancers less than 1 cm in diameter. They have very excellent prognosis although, those presenting with distant metastasis requires more aggressive therapy.

Complications

1. Spread.
2. Other complications:
 - a) Local (infiltration of surroundings):
 - Recurrent laryngeal n. → hoarseness of voice.
 - Sympathetic chain → Horner's \$.
 - Trachea → dyspnea not related to posture.
 - Esophagus → dysphagia.
 - b) General:
 - Cachexia
 - Metastasis (jaundice, cough, hemoptysis, pleural effusion)

Staging

De Groot staging (Philadelphia 1989):

- Stage I** → Tumor with single or multiple intra-thyroid foci.
- Stage II** → Tumor with mobile neck LN.s
- Stage III** → Tumor with fixed neck LN.s ($\pm$ local invasion).
- Stage IV** → Tumor with distant metastasis.

Grading

- **G 1** → Well differentiated
- **G 2** → moderately differentiated
- **G 3** → poorly differentiated

Clinical Picture

⇒ Type of patient:

- Female 15 years old, thyroid swelling in neck, LN (papillary carcinoma).
- Female 40 years old, rapid progressive swelling in neck and sometimes skull metastasis.
- Female old age complaining of rapid progressive swelling in neck with hoarseness of voice.

⇒ Typical presentation:

Symptoms:

- 1- **Swelling** in lower part of front of the neck → rapidly growing (especially in anaplastic carcinoma).
- 2- Early painless & late **painful** & pain may referred to ear (along auricular branch of vagus "Arnold nerve")
- 3- **Infiltrative** manifestations: dyspnea, dysphagia, hoarseness of voice & Horner's \$.
- 4- **Metastatic** manifestations: neck masses, skull swelling, hemoptysis & jaundice.

Signs:

❖ General examination:

- Cachexia.
- metastasis (skull metastasis , jaundice , ascites)

❖ Local examination:

A- Thyroid swelling :

1. **Early** → mobile thyroid lump.
2. **Late** → Hard fixed infiltrate the surrounding structures

B- Neck LNs: May be enlarged & hard (mobile or fixed to skin & m.)

C- Surroundings:

- 1- **Trachea** → is fixed to the gland
- 2- **Carotid artery** → Absent carotid pulse (Berry's sign). (Infiltration of carotid sheath)

⇒ Occasional presentation:

- 1- Enlarged neck LNs while thyroid carcinoma is not palpable, it occurs in papillary carcinoma (occult carcinoma).
- 2- Solitary skull metastasis (it occurs in follicular carcinoma)
- 3- Histopathological surprise: it may be seen in follicular carcinoma (During subtotal thyroidectomy for SNG → histopathology → follicular carcinoma). Histo-pathological surprise can be avoided by good history (sudden change of characters)

DD

- 1- Other causes of solitary thyroid nodule.
- 2- Riedel's thyroiditis.
- 3- Calcified nodular goitre.

Investigations

Investigations

For diagnosis

For staging

For preoperative preparation

For Follow up

For screening

A. For diagnosis:1. **Lab:**

Thyroid function → normal; however TSH may be raised.

2. **Imaging:**

a) U/S:

Cystic → aspiration (Criteria of malignancy)

Solid → FNABC

U/S is more likely sensitive to identify small malignant LNs

b) Isotope scanning (**of limited diagnostic value**): cold nodule but extremely rare hyperfunction → thyrotoxicosis.

3. **Biopsy:**

a) FNABC

- 90 % accuracy, simple, rapid & inexpensive.
- Often causes (hemorrhage, cyst.....etc)
- Can't differentiate between follicular adenoma and follicular carcinoma because FNABC can't detect capsular and blood invasion

b) Hemithyroidectomy and paraffin section in follicular neoplasia

c) In an anaplastic & obviously irremovable carcinoma, incisional or core needle biopsy is justified.

B. For staging:

1. CT scan, MRI, U/S → to detect direct spread
2. CXR → lung metastasis.
3. Bone scan → bone metastasis (not done except after total thyroidectomy).
4. Abdominal U/S → liver metastasis.
5. Direct laryngoscopy, bronchoscopy, esophagoscopy.

C. For pre-operative preparation

CBC, FBS, KFTs, LFTs, CXR, ECG

D. For follow up:1. **Tumor markers:**

- Calcitonin in medullary carcinoma.
- Thyroglobulin in differentiated follicular carcinoma.

2. **Positron emission tomography (PET)**

- Indication: in follicular carcinoma treated with thyroidectomy with post-operative rise of thyroglobulin level

Treatment

Treatment

Prophylactic treatment

Treatment of tumor

Treatment of complications

Follow up

Prognosis

TTT of special problems as Histo-pathological surprise

1. Treatment of the tumor:

A- Differentiated carcinoma (Papillary & follicular):

I- Surgery:

	Radical approach
Definition	Total Thyroidectomy + central node dissection + thyroxine replacement
Indications	Preoperative diagnosis of high risk cancer or bilateral disease. There is a clear indication for postoperative radioiodine therapy.
Advantages	↓ Local recurrence Facilitate postoperative use of radioiodine to detect metastasis.
Disadvantages	Hypoparathyroidism Recurrent nerve injury Hypothyroidism

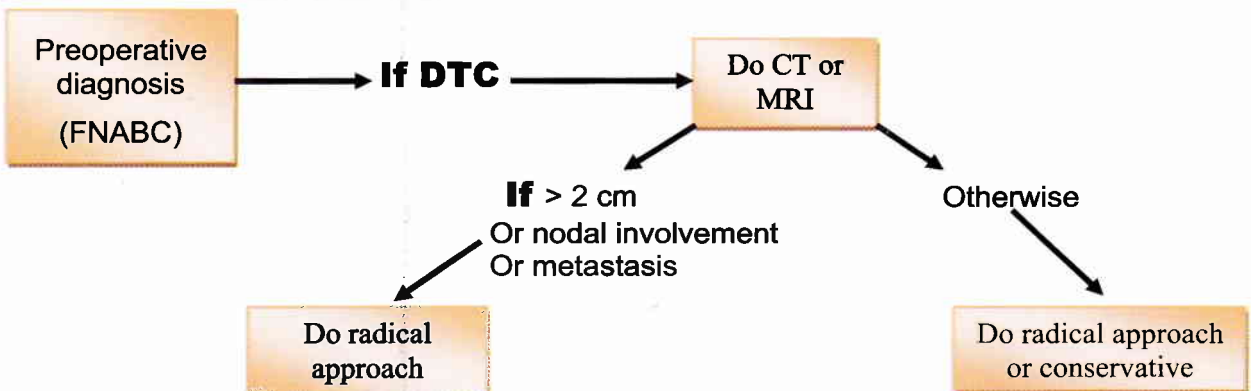
Management of LNs:

- In children → no prophylactic block dissection of LNs.
- In adults → prophylactic block dissection of central group of LNs is done.
- If one LN is affected block dissection of LNs in the neck is done.
- In the past: cherry pick dissection of LNs was done.

Conservative approach

- there is other opinion that small cancers confined to one lobe can be managed by (lobectomy + TSH suppression)

Authors current practice :



2- Radioactive iodine:

➤ Aim:

- 1- Destroy remnants of normal thyroid tissue if present.
- 2- To ablate any metastasis from the tumor.

➤ Methods:

- After total thyroidectomy we wait till manifestations of myxedema appear
 - Pre-therapy scan: small dose of radioactive iodine is given and total body scan is done.
 - If there is metastasis: large ablative dose of radioactive iodine is given.
 - Post-therapy scan: done after the ablative dose by few weeks.

B- Anaplastic carcinoma:

- 1- Unfortunately, the majority of cases are irresectable at time of presentation
 - Tracheostomy or isthmus resection (or surgical debulking).
 - **Radiotherapy and chemotherapy** (down staging).
- 2- Rare cases are operable → the tumor should be excised as completely as possible (total thyroidectomy) and then treated by radiation and chemotherapy.

2. Treatment of complications:

- 1) Tracheostomy for tracheal infiltration.
- 2) Gastrostomy for esophageal infiltration.
- 3) Isolated metastatic deposits of follicular and papillary carcinoma should be surgically removed and treated with I^{131} after total thyroidectomy or thyroid ablation with radioactive iodine.

3. Post operative follow up:

- 1) Every 3 month by thyroid scanning, clinical examination and tumor marker (it is helpful to measure serum level of thyroglobulin which are usually increased > 2 ng/ml in patients with residual tumor after total thyroidectomy).
- 2) Aim:
 - To detect local spread.
 - Distant metastasis.
 - Post operative complications as myxedema and RLN injury.

4. Prognosis:**➡ Certain factors increase the risk including:**

- a) Age: males above 40 & females above 50 years of age.
- b) Size of the lesion if more than 5 cm in diameter.
- c) Presence of capsular or blood vessel invasion.

Medullary Carcinoma (6%)

Origin

It arises from **para-follicular "C" cells** that secrete **calcitonin**.

Incidence

- Old age
- 6%

Etiology

- a- Sporadic type : no specific age or sex incidence & it is fatal
 - b- Familial : as in MEN-II (Sipple's syndrome) → (medullary carcinoma, pheochromotoma, parathyroid adenoma)
- N.B They have the same embryological origin*

Pathology

- **Macro** → The lesion may be solitary or multiple.
- **Micro** → sheets of anaplastic cells in amyloid stroma (Amyloid substance in stroma (stained by congo-red stain and seen by polarizing microscope))
- **Spread** → **Lymphatic** spread (50%) (**Mediastinal LN** involvement can occur in medullary carcinoma)
Blood spread (mainly to liver)

Clinical picture

Symptoms:

- 1- **Swelling** in lower part of front of the neck → Rapidly growing
- 2- Early painless & late **painful** & pain may referred to ear (along auricular branch of vagus "Arnold nerve")
- 3- **Infiltrative** manifestations: dyspnea, dysphagia, hoarseness of voice & Horner's \$.
- 4- **Metastatic** manifestations.
- 5- **Others** → Secretion of 5-HT (serotonin) → carcinoid syndrome (diarrhea, bronchospasm and flushing)

Signs:

- ❖ **General examination:**
 - Cachexia.
 - metastasis (skull metastasis , jaundice , ascites)
- ❖ **Local examination:**
 - a- **Thyroid swelling :**
 1. **Early** → mobile thyroid lump.
 2. **Late** → Hard fixed infiltrate the surrounding structures
 - b- **Neck LNs:** May be enlarged & hard (mobile or fixed to skin & m.)
 - c- **Surroundings:**
 - 1- **Trachea** → is fixed to the gland
 - 2- **Carotid artery** → Absent carotid pulse (Berry's sign). (Infiltration of carotid sheath)

Investigations

- **For screening:** In medullary carcinoma (familial type) → calcitonin, calcium → if high → total thyroidectomy even if thyroid is normal by other investigations
- **For diagnosis:** US, FNABC, ↑ serum calcitonin > 0.08 ng /ml (the level falls after resection of the tumor)

- For staging:
 - CT scan.
 - CXR → lung metastasis.
 - Bone scan → bone metastasis
 - Abdominal U/S → liver metastasis.
 - Direct laryngoscopy, bronchoscopy & esophagoscopy.
- For pre-operative preparation:
 - CBC, FBS, KFTs, LFTs, CXR, ECG
- To rule out pheochromocytoma → urinary catecholamines

N.B Medullary carcinoma is characterized by Secretion of calcitonin which can be used as a tumor marker in patient relatives, if high → total thyroidectomy (prophylactic thyroidectomy)

Treatment:

- Thyroid → Total thyroidectomy (removal of both lobes and isthmus of thyroid gland) + resection of metastatic L.N.s.
- Lymph nodes management →
 - Medullary carcinoma is associated with high incidence of nodal involvement.
 - Central neck node dissection should be done in all patients.
 - Modified radical neck dissection for primary tumor > 1.5 cm in diameter and when nodes are involvement
- Parathyroid → Sporadic type (preserve all parathyroid gland)
→ Familial type, we preserve only 1/2 parathyroid gland (for fear of hyperparathyroidism).

N.B If familiar type → treatment of pheochromocytoma is done at first by (combined alpha & beta blockers) then adrenalectomy.

No rule of radioactive iodine as C-cells can not pick up iodine

Prognosis

- Moderately malignant – Not TSH dependant
- Prognosis is better in sporadic type than familial type.

Thyroid Lymphoma (4%)

In the past, many malignant lymphomas were diagnosed as small round cell anaplastic carcinoma

- Especially non Hodgkin.
- May be Burkitt's lymphoma.
- May be on top of Hashimoto.
- 1st extra-nodal (if 2nd to nodal → bad prognosis).

Treatment

- Radiotherapy and chemotherapy.
- Highly radiosensitive & of good prognosis.

ORAL QUESTIONS

APUD cells: these are cells derived from the neural crest (neuroendocrine cells) & scattered all over the body, they have capability to synthesize vasoactive intestinal polypeptide (VIP) & in doing they utilize the enzyme amine precursor uptake & decarboxylase (APUD).

MEN-I is characterized by 3Ps:

- ① Pituitary tumor.
- ② Parathyroid adenoma.
- ③ Pancreatic tumor.

MEN-II is of 2 types (MEN= multiple endocrinal neoplasm)

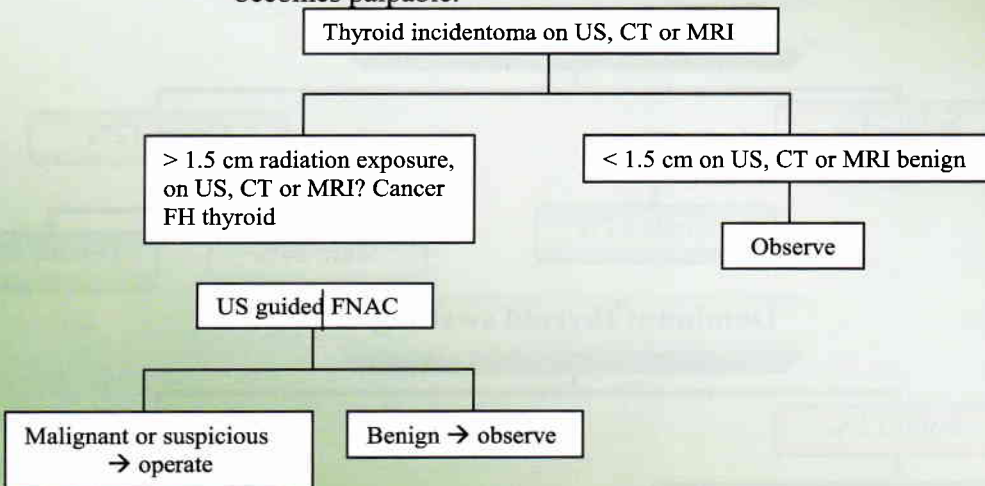
A: of good prognosis
 Medullary carcinoma
 Pheochromocytoma
 Parathyroid Adenoma.

B: (of bad prognosis).

As A + Neurofibroma. + Hirschsprung + Marfanoid features

Thyroid Incidentaloma:

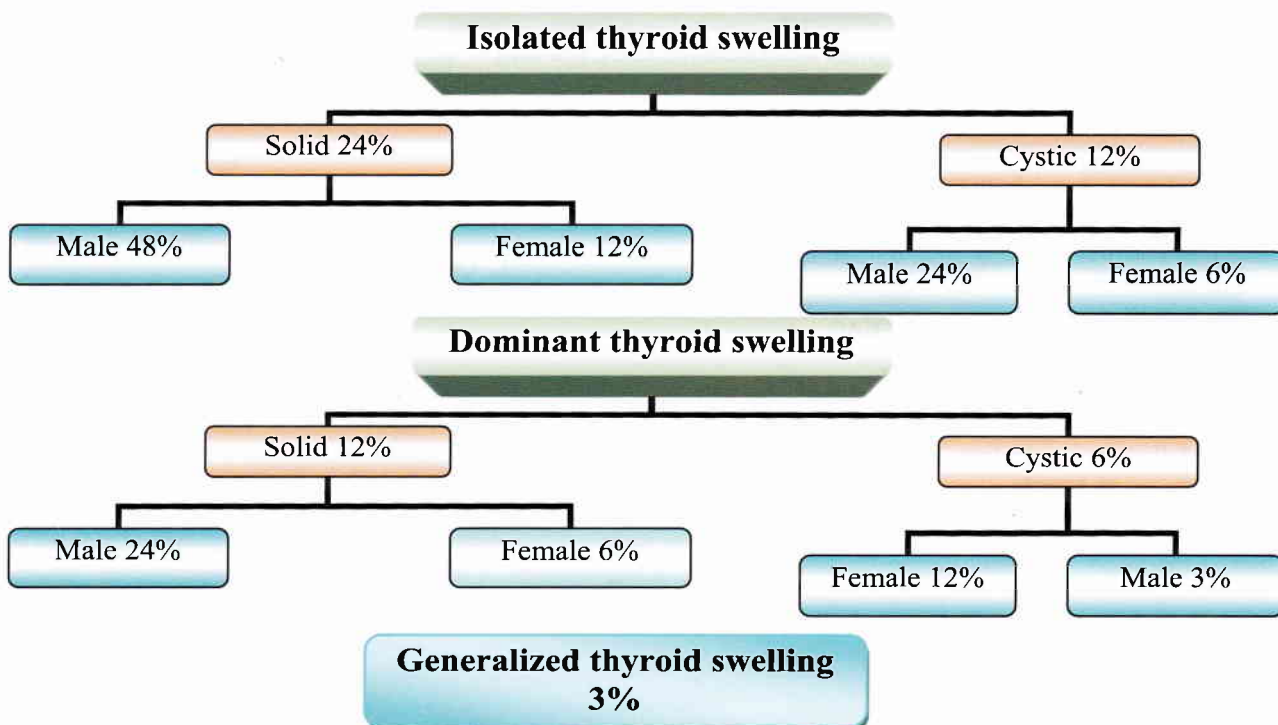
- **Definition:** These are clinically unsuspected & impalpable thyroid swelling.
- **Cause:** the term thyroid incidentaloma has been created due to ↑ use of imaging modalities for non-thyroid pathology.
- **Management:** The majority of impalpable thyroid swelling can be managed by single annual review with no intervention, unless certain criteria be met or the swelling becomes palpable.



TNM staging of thyroid cancer:

- Tumor:
 - Tx → primary can not be assessed.
 - T0 → no evidence of lry.
 - T1 → limited to thyroid, 1 cm or less.
 - T2 → limited to thyroid > 1cm but < 4cm.
 - T3 → limited to thyroid > 4cm.
 - T4 → extending beyond capsule, any size.
- Nodes:
 - Nx → can not be assessed.
 - N0 → no regional node metastasis.
 - N1 → regional node metastasis.
- Metastasis:
 - Mx → can not be assessed.
 - M0 → no metastasis.
 - M1 → metastasis present.

Stage	Under 45 years	Over 45 years
I	Any T, any N, M0	T1, N0, M0
II	Any T, any N, M1	T2, N0, M0 or T3, N0, M0
III		T4, N0, M0 or any T, N1, M0
IV		Any T, any N, M1

Rule of 12**Risk of malignancy in the thyroid swelling can be expressed as rule of 12**

The risk is greater in :

- 1- Isolated > Dominant
- 2- Solid > Cystic
- 3- Male > Female

Solitary Thyroid Nodule (STN)

➔ See differential diagnosis book.

Recurrent Goitre

Incidence of recurrence is 5-15% after 10 years from the operation.

Types of Recurrent Goitre

False recurrence It is due to

Foreign body reaction, a granuloma around silk ligature or small piece of gauze left accidentally.

Inadequate initial removal.

- **Partial** → leaving part equal to normal lobe size on each side (4x2.5x2 cm).
- **Subtotal** → leaving a part less than the normal lobe (in simple goitre leave 1/5 of normal lobe on each side).
- **Toxic** → leave 1/8 of normal lobe on each side (**subtotal**).

Non removal of certain part of the gland

1. Isthmus.
2. Pyramidal lobe → if present should removed completely.

True recurrence

Commonly due to persistence of the same etiological factors of the primary goitre.

- a) Iodine deficiency
- b) Dyshormonogenesis.
- c) **Neoplastic**

Lack of post-operative administration of T3 & T4 in SNG (thyroid hormones keep post-operative TSH not fluctuating.

▪ **Indications for operation of thyroid swelling**

- Neoplasia:
 - FNAC +ve.
 - Clinical suspicious including: age, male sex, hard texture, fixity, recurrent laryngeal nerve palsy and lymphadenopathy.
- Recurrent cyst.
- Toxic adenoma.
- Pressure symptoms.
- Cosmiosis.
- Patient's wish.

CHAPTER 3

OTHER ENDOCRINAL DISORDERS

Parathyroid Gland

Anatomical Notes

- 4 in number (80%) or 3 in number (13%) or 5 in number (6%).
- Small, oval, mobile, yellowish.
- 0.5 cm in diameter.
- Related directly to thyroid gland.



Hyperparathyroidism

Etiology

1. 1ry Hyperparathyroidism:

a- Adenoma:

- 92 % of cases of hyperparathyroidism.
- Middle aged female.
- Usually affect one gland.

b- Hyperplasia:

- 3 % of cases.
- Affect 4 glands.

c- Carcinoma:

- Very rare <1 %.
- PTH & Ca are elevated.

d- Multiple adenomatosis (4 %)

2. 2ry Hyperparathyroidism (Normocalcemic Hyperthyroidism):

- Due to compensatory hyperplasia of parathyroid gland due to prolonged hypocalcemia e.g : CRF & malabsorption
- PTH is high before Ca level is low or normal

3. Tertiary Hyperparathyroidism:

- After prolonged 2ry hyperparathyroidism , secretion of PTH becomes autonomous
- PTH & Ca levels are elevated.

4. Paramalignant Syndrome:

- Ectopic secretion of PTH by small cell bronchial carcinoma

Clinical Picture

*(Commonest presentation is asymptomatic hypercalcemia)
(Bone + stone + psychic moan + abdominal groan)*

1. Bone:

- Bone pain.
- Osteitis fibrosa cystica → multiple bone cysts.
- Pathological fractures.

2. Stone:

- Recurrent renal stones.
- Nephrocalcinosis: calcification in renal parynchema.

3. GIT (abdominal groan):

- Anorexia, nausea & vomiting.
- Peptic ulceration.
- Acute pancreatitis.

4. Psychic moan:

- Common in women.
- Apathy, lack of concentration.
- Depression.

In patients below 60 yrs urological presentation are common, while above 60 yrs neuromuscular & psychiatric manifestations dominate

DD of Hypercalcemia

- Hyperparathyroidism.
- Cancer with endocrine secretion.
- Multiple myeloma.
- Thyrotoxicosis.
- Drugs: e.g. Thiazide diuretics, Lithium.
- 2ry tumor in bones.
- Vit. D intoxication.

Investigations

- ⇒ **Investigation for hyperparathyroidism :**
 - ↑ Serum calcium except in 2ry hyperparathyroidism (normocalcemic hyperparathyroidism)
 - ↓ Serum Phosphorus.
 - ↑ Serum PTH.
 - X-ray bone: multiple bone cysts & resorption of terminal bone of phalanx.
- ⇒ **Investigations for localization in recurrent cases:**
 - CT scan.
 - Thallium 201, TC₉₉
 - Subtraction scan (old).
 - Sesta-MIBI scanning (most accurate, better than U/S).

Treatment

- **1ry : (surgical):** Surgery to remove the enlarged gland (or glands) is the main treatment for the disorder and cures it in 95 percent of operations.
 - Adenoma : resects it
 - Hyperplasia :remove 2/3 gland & implant 1/2 in deltoid arm
- **2ry (medical)**
 - 1 alpha hydroxyl vit. D3 → ↑ Ca absorption → -ve feedback on parathyroid gland.
 - **Calcimimetics** are a new class of drug that turns off secretion of PTH. They have been approved by the Food and Drug Administration for the treatment of hyperparathyroidism secondary to kidney failure with dialysis, and primary hyperparathyroidism caused by parathyroid cancer. They have not been approved for primary hyperparathyroidism, but some physicians have begun prescribing calcimimetics for some patients with this condition.
- **Tertiary:**
 - Total parathyroidectomy with autotransplantation of parathyroid fragment equal to normal size in arm ms to be easily located if a problem persists.

Hypoparathyroidism

Definition

- Rare condition characterized by hypocalcaemia due to reduced production of parathormone.

Causes

- Post thyroid or parathyroid surgery.
- Idiopathic.
- Pseudohypoparathyroidism (reduced sensitivity to parathormone).

Pathology

- Reduced calcium increases neuromuscular excitability.

Clinical Picture

- Perioral paresthesia, cramps, tetany.
- Chvostek's sign: tapering over facial nerve induce facial muscle contractions.
- Trousseau's sign: inflating blood pressure cuff to above systolic pressure induce typical main d'accoucheur or carpopedal spasm.
- Prolonged QT interval on ECG.

Diagnosis

- Calcium and parathormone levels decreased.

Treatment

- Calcium and vitamin D.

Myxedema

Etiology

- 1- **Idiopathic atrophy of the thyroid gland:** thyroid antibodies are present in 80% of cases.
- 2- **Hashimoto's disease:** thyroid antibodies are present in 100 % of cases.
- 3- **Endemic myxedema:** due to prolonged iodine deficiency.
- 4- **Iatrogenic:** due to thyroidectomy, radioactive iodine deficiency or prolonged use of Antithyroid drugs.
- 5- **Secondary myxedema:** due to pituitary or hypothalamic disease.

Incidence

- 1- **Age:** more common between 30 & 50 years.
- 2- **Sex:** more common in females.

Clinical Picture

A- General Manifestations:

- 1- Intolerance to cold and subnormal body temperature.
- 2- Fatigue, weakness, slow movement.
- 3- Weight gain.
- 4- Eyebrows are sparse with fall of hair over the outer third.

B- Cutaneous Manifestations:

- 1- Dry, coarse, pale & cold skin
- 2- Sweating is absent and hair is sparse and brittle.
- 3- Skin is thickened and does not pit on pressure.
- 4- Yellowish skin due to carotinemia.

C- Cardiovascular manifestations:

- 1- Sinus bradycardia.
- 2- Atherosclerosis.
- 3- Hypertension.

D- Neurological manifestations:

- 1- Mental impairment. Sluggish movements, slow thinking and poor memory.
- 2- Peripheral neuropathy.
- 3- Delayed relaxation of tendon jerks (suspended jerks)

E- Gastrointestinal Manifestations:

Constipation due to decreased intestinal motility.

F- Genital Manifestations:

- 1- Menorrhagia in females.
- 2- Gynaecomastia in males.

Investigations

1- Thyroid function tests:

- 1- T₃, T₄ (free and total hormones) are reduced.
- 2- TSH is increased in primary and decreased in secondary hyperthyroidism.

2- Biochemical changes:

- 1- Serum cholesterol level is high.
- 2- Glucose tolerance curve is flat.

3- Blood Picture:

anemia.

DD

- 1- Nephritic syndrome.
- 2- Obesity.
- 3- Depression.

Treatment

⇒ Replacement therapy with L-thyroxine:

- Started in a dose of 50 microgram/day.
- The dose is increased every 2 weeks by 50 microgram.
- The average maintenance dose is 200 microgram/day.
- Therapy is monitored by TSH level (most important), T₃ & T₄, cholesterol level normalization of ECG and clinical improvement.

Diseases of the adrenal cortex

1. Chronic hypocortisolism (Addison's Diseases)

Causes

1. Autoimmune.
2. Amyloidosis.
3. Tuberculosis.
4. Metastatic carcinoma.
5. Surgical removal of the adrenals.

Clinical Picture

- Muscles weakness.
- ↓ blood pressure.
- Pigmentation of the skin.

Treatment

- 20-30 mg hydrocortisone in divided doses with fludrocortisone. (0.1 mg daily).

2. Cushing's syndrome

Definition

Cushing's syndrome is a condition caused by chronic exposure to elevated levels of the hormone cortisol (hypercortisolism). When a pituitary tumor is the cause of high cortisol, the disorder is called Cushing's disease

❖ Age & sex:

- Relatively rare
- Commonly affects adults aged 20 to 50. - ♀ : ♂ = 3 : 1

Etiology

I. ACTH-dependant:

1- Pituitary adenoma:

- It secretes excess ACTH leading to bilateral adrenal hyperplasia (Cushing's disease). This represents about 80% of non-iatrogenic cases.

2- Ectopic ACTH syndrome:

- Paramalignant syndrome e.g. small cell bronchial carcinoma.

3- Iatrogenic:

- Prolonged ACTH therapy (cushinoid syndrome)

II. Non-ACTH-dependant:

1. Adrenal tumor: adenoma or carcinoma.
2. Iatrogenic: Prolonged corticosteroid therapy (cushinoid syndrome)

Clinical Picture *(Common signs and symptoms of Cushing's)*

I. Fat redistribution:

- Patients may have increased adipose tissue:
 - o in the face (**moon facies**)
 - o Upper back at the base of the neck (**buffalo hump**)
 - o Above the clavicles (**supraclavicular fat pads**).
- Trunkal obesity with pendulous abdomen
- Increased waist to hip ratio greater than 1 in men and 0.8 in women

II. Skin

- Facial plethora (red cheeks).
- Red striae, common over the abdomen, buttocks, back, upper thighs, upper arms, and breasts.
- Ecchymosis.
- Cutaneous atrophy and thinning of skin may be evident.
- Hirsutism and male pattern hair may be present in women.
- Increased facial hair.
- Poor wound healing.

III. Cardiovascular and renal

- Hypertension.
- Volume expansion with edema.

IV. GIT

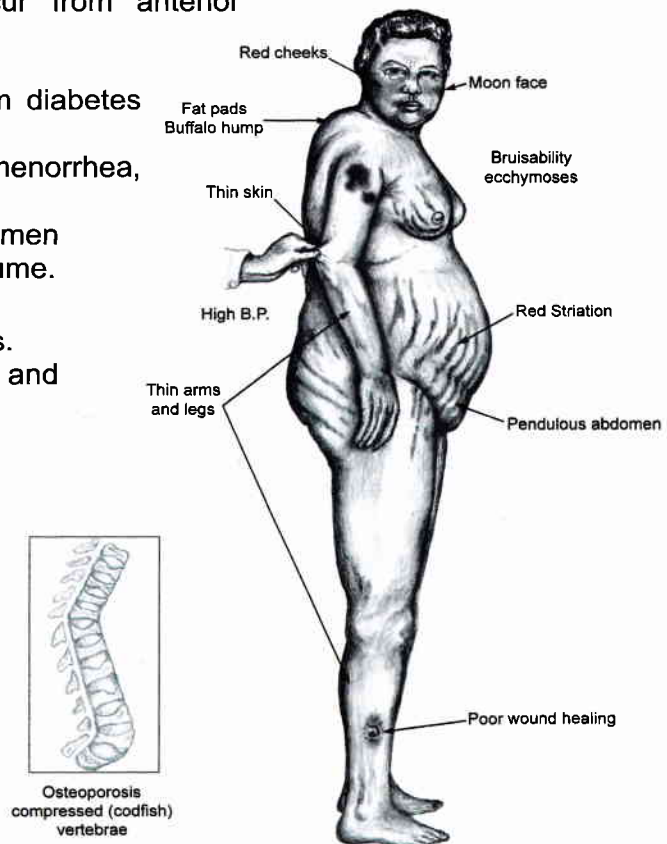
- Peptic ulceration.

V. Endocrine

- Diabetes mellitus.(steroid induced)
- Hypothyroidism may occur from anterior pituitary tumors.
- Galactorrhea.
- Polyuria and nocturia from diabetes insipidus.
- Menstrual irregularities, amenorrhea, and infertility.
- Low testosterone levels in men
→decreased testicular volume.

VI. Skeletal/muscular

- Proximal muscle weakness.
- Osteoporosis→fractures and kyphosis
- Bone pain.



DD

- Other causes of obesity, hypertension & DM especially when combined.
- Other causes of hirsutism.
- Differential diagnosis of the cause: pituitary, adrenal or ectopic.

Investigations**A. Investigations for diagnosis of Cushing's syndrome:**

- **↑Urinary free cortisol or 17-hydroxycorticosteroids:**
- **Plasma cortisol level:**
 - Normal 5-20 mg/dl & there is a normal circadian rhythm
 - In early stages of Cushing's syndrome there is loss of circadian rhythm.
 - In later stages there is persistent elevation of cortisol level.
- **Low-dose dexamethasone suppression test:**
 - Dexamethasone 0.5 mg/6 hours is given for 2 days.
 - In normal individuals this causes inhibition of ACTH secretion by negative feedback mechanism so cortisol level in blood will decrease.
 - In Cushing's syndrome there will be no change in cortisol level.

⇒ Less important investigations:

- **Insulin tolerance test:**
 - Hypoglycemia stimulates an increase in plasma cortisol in normal individuals but not in patients with Cushing's syndrome.
- **Blood picture:**
 - Increased red cells & neutrophils.
 - Decreased lymphocytes & eosinophils.
- **Blood chemistry:**
 - Increased Na & glucose.
 - Decreased K.

B. Investigations for diagnosis of the cause:

- **Plasma ACTH:**
 - Normal level: 10-80 ng/liter.
 - Low level in adrenal tumors.
 - High level in ACTH-dependent cases and ectopic ACTH secretion.
 - If not available shift to high dexamethasone suppression test.
- **High-dose dexamethasone suppression test:**
 - Dexamethasone 2 mg/6 hours is given for 2 days.
 - Decrease in plasma cortisol occurs only in pituitary tumours but not in adrenal tumours or ectopic ACTH syndrome.
- **Diagnosis of pituitary tumours:**
 - MRI & CT scan.
 - Inferior petrosal sinus sampling.
- **Diagnosis & localization of adrenal tumours :**
 - U/S, CT scan, MRI or selective adrenal vein sampling.

Treatment**1- For pituitary tumours:**

- 1- Trans-sphenoidal removal of the tumor (treatment of choice)
- 2- Hypophysectomy or pituitary irradiation followed by replacement therapy

2- For adrenal tumours:

- 1- Surgical removal of the tumour.
- 2- This should be followed by suboptimal replacement therapy with low-dose steroids till the other atrophic adrenal gland recovers from suppression.

3- For paramalignant syndrome: treatment of the primary tumor if possible.**4- Medical therapy:**

- Used to prepare patients before surgery or if surgery is contraindicated.
- Includes drugs that inhibit cortisol production e.g. metyrapone)

Adrenal gland

Anatomy

- There are adrenal glands on each side, lying immediately above the kidney
- each gland weight 4:6 gm

Arterial Supply

- Superior suprarenal from phrenic artery.
- Middle suprarenal from aorta.
- Inferior suprarenal from renal a.

Venous Drainage

- Left supra renal v. to left renal v.
- Right supra renal v. to IVC

Histology & Physiology

- The gland is formed of cortex and medulla.
- The cortex is formed of 3 zones: zona grnulosa secrete mineralocorticoid controlled by renin-angiotensin system
- Zona fasciculata secrete glucocorticoid controlled by ACTH.
- Zona reticularis secretes androgen.
- The adrenal medulla secretes catecholamines.
- Chromaffin cells is a part of neuroendocrinal cell or APUD system so this tumor either solitary or a part of (MEN) syndrome.

Pheochromocytoma

Definition

- A tumor of chromaffin tissue secreting catecholamines.
- **Types:**
 - Sporadic: usually in adult, 10% malignant< 10% bilateral, 10% multiple.
 - Familial: usually in children 50% bilateral, may be a part of (MEN) type 2.

Clinical Picture

Early Hypertension or DM in young age

- 1- Hypertension which is usually paroxysmal.
- 2- Attacks of sympathetic over activity (hypercatecholaminaemia):
 - Tachycardia & palpitation.
 - Sweating & pallor.
 - Anxiety & tremors.
 - Arrhythmia & precipitation of angina.
- 3- Hyperglycemia.
- 4- Hypertrophic cardiomyopathy may occur.
- 5- May be associated features of multiple endocrine neoplasia (MEN) e.g.
 - Medullary thyroid carcinoma.
 - Hyperparathyroidism.
 - Neurofibromatosis.

Investigations

I. Laboratory:

- 1- Increased urinary vanillylmandelic acid (VMA) (N: 2-7 mg/24 hrs).
- 2- Increased urinary catecholamines.
- 3- Increased plasma catecholamines.

2. Radiological

- 1- Abdominal ultrasonography
- 2- CT scan or MRI
- 3- Selective adrenal vein sampling.

Treatment

- **Adrenalectomy** of the diseased side after preoperative preparation
- **Preoperative preparation:** alpha blockers 7:10 days followed by beta blockers 3:4 days or combined alpha and beta.
- **Operative:** anesthesia by nitrous oxide

NB. Avoid halothane to avoid arrhythmia

▪ **Approaches of adrenalectomy:**

1. Posterior approach (lumbar approach).
2. Thoracoabdominal approach (bed of 10th rib).
3. Anterior approach (for bilateral exposure).
4. Laparoscopic adrenalectomy.

➡ The organ of zuckerKandl is formed of embryonic chromaffin cells around the abdominal aorta. These cells normally become atrophic during childhood, but are major sites of extra-adrenal pheochromocytoma.

➡ **Indications of screening hypertensive patients for Pheochromocytoma:**

1. Recent onset with retinopathy or DM.
2. Symptomatic HTN with vasomotor phenomena or DM.
3. Episodic HTN especially with postural HTN.

CHAPTER: 4

SWELLINGS & CYSTS

- 1- General scheme for swellings
- 2- Lipoma
- 3- Tumors of the nerves
- 4- Hamartomas
- 5- Classification of cysts
- 6- Sebaceous cysts
- 7- Dermoid cysts
- 8- Thyroglossal cysts
- 9- Branchial cysts
- 10- Ganglion
- 11- Cystic hygroma
- 12- Laryngocele
- 13- Penumatocele
- 14- Cysts of the floor of the mouth
- 15- Keloid
- 16- Baker's cyst
- 17- Desmoid tumor
- 18- Bursae
- 19- Carotid body tumor
- 20- Congenital Torticollis

Swellings

- It is a common surgical complain.
- We can classify swellings according to:
 1. **Consistency:**
 - a. Solid swellings.
 - b. Cystic swellings.
 2. **Sites:**
 - a. Swellings of the face.
 - b. Swellings of the hand.
 - c. Swellings of the neck.
 3. **Origin:**
 - a. Skin.
 - b. Subcutaneous tissue.
 - c. Muscle.
 - d. Nerve.
 - e. Blood vessel.
 - f. Bone.
 - g. Visceral organ.

Scheme for diagnosis of any swelling

Definition

About the nature of the swelling:

- Tumor.
- Hamartoma.
- Cyst.

Incidence

- It is common, rare, and very rare.
- Age of incidence.
- Sex of incidence.

Etiology

- Congenital – Embryology – or acquired.

Pathology

Origin

Site

- Commonest it ...
- Less common in ...
- Never in ...

Macroscopic Picture

- | | | |
|------------|----------------|-----------------|
| □ Shape. | □ Color. | □ Number. |
| □ Size. | □ Consistency. | □ Blood Supply. |
| □ Surface. | □ Cut section. | □ Capsule. |

Microscopic Picture

Types

Diagnosis

Clinical Picture

- **Age:**
- **Complaint:** - *Swelling*:
 - Painless or painful.
 - Slowly or rapidly growing.
 - Site.
- **Signs:**
 - General: according to nature of the swelling.
 - Local:
 - ◆ **Inspection:**
 1. Site and number.
 2. Shape and size:
 - ▲ Rounded. ▲ Oval. ▲ Irregular.
 3. Skin overlying.
 4. Special sings.
 - ◆ **Palpation:**
 1. Surface:
 - ▲ Smooth. ▲ Lobulated.
 2. Edge:
 - ▲ Well defined ▲ Ill defined. ▲ Pedunculated.
 3. Consistency:
 - ▲ Cystic:
 - Paget's test. - Fluctuation test.
 - ▲ Solid:
 - Soft - Firm. - Hard.
 4. Mobility:
 - ▲ Freely mobile.
 - ▲ From side to side.
 - ▲ From above downward.
 - ▲ Restricted.
 5. Signs of inflammation: "Hotness, redness, tenderness, and edema"
 6. Special sings:
 - ▲ Pulsation: vascular swelling.
 - ▲ Compressible: hemangiomas.
 - ▲ Reducible.
 - ▲ Transillumination: cyst with clear fluid.
 7. Draining L.Ns:
 - ▲ Enlarged.
 - ▲ Not enlarged.
 - ◆ **Percussion:** not important.
 - ◆ **Auscultation:** important in vascular swelling.

Complication

DD

From common swelling related to site.

Investigations

- Specific.
- Routine.

Treatment

According to the nature of the swelling.

Lipoma

Definition

- It is a benign tumor arising from adipose tissue.

Pathology

Pathological (structural) Classification

- Lipoma is classified according to structure into:
 - Pure lipoma: mainly adipose tissue (non compressible).
 - Fibrolipoma: with extensive fibrous tissue (firm).
 - Hemangiolipoma or navio lipoma: with extensive vascular tissue (partially compressible as blood vessels are compressible while fat is not).
 - Myxolipoma (myxematous tissue)

- **Types:** commonest is s.c.
- **C/P:** swelling, pain, comp.
- **Comp.:** as cyst + liposarcoma
- **Invest.:** biopsy if needed, imaging if deep
- **TTT:** excision

Gross picture

- Size:** variable.
- Shape:** oval or rounded.
- Surface:** Lobulated.
- Consistency:**
 - Always soft.
 - Except, Firm in → Fibrolipoma (due to fibrous tissue elements).
→ Subfascial lipoma (as it is under tension).
- Color:** Yellowish.
- Cutsection:** thin fibrous capsule sends fibrous tissue septa divide the tumor into multiple spaces.
- Capsule:** 2 capsules:
 - ◆ Inner true: Formed of fibrous tissue.
 - ◆ Outer false: Formed by compressed surrounding structures.
 - ◆ Between the 2 capsules there is a line of cleavage that facilitates enucleation of the tumor.
 - ◆ Lipoma can be classified according to presence or absence of the capsule into: encapsulated or diffuse.
- Origin:** adipose tissue.
- Blood supply:** through the pedicle at one side of the tumor.
- Number:**
 - Solitary well defined swelling .
 - Multiple lipomatosis is when the limbs or the trunk are the seat of multiple lipomas.
 - Diffuse lipomatous deposits: this can occur in certain areas e.g.: patient with myxedema has supraclavicular fatty deposits, elderly persons may develop lipomatous deposits below the chin and some times females may develop painful fatty deposits in the thigh (Dercum's disease)

Microscopic Picture

- Aggregation of **fat cells** with signet ring appearance. separated by **fibrous connective tissue** stroma containing **blood vessels** arising from the pedicle..

Clinical Picture

- It differs according to the site.

Complaint

- Painless slowly growing swelling.
- Cosmetic disfigurement.
- Painful lipoma (neurolipoma, Decrum, Complicated lipoma)



Signs

- ▣ **General:** e.g. fever if lipoma is complicated by infection.
- ▣ **Local:** According to site:

1. Subcutaneous lipoma

- ◆ The commonest type
- ◆ Common in back, shoulder, buttocks, forehead and limbs.
- ◆ Lobulated painless mass **attached** to skin at multiple points
- ◆ **Soft** in consistency but may be firm if excess fibrous tissue in it. Sometimes may give **pseudo-fluctuation** due to high mobility of tumor in its bed and some degrees of fat liquefaction inside it.
- ◆ Has well defined **slippery edge** due to double capsule.
- ◆ May be solitary & multiple.
- ◆ May be pedunculated → lipoma arborescens
- ◆ Mobile over deep structures.



Lipoma of the arm

2. Subfascial lipoma

- ◆ **Firm**
- ◆ **Not attached to skin.**
- ◆ **Doesn't have a slippery edge.**
- ◆ Occurs under deep fascia e.g. palmar or plantar fascia or in the areolar layer in the epicranial aponeurosis, difficult to diagnose.
- ◆ Increased incidence in forehead.

3. Sub synovial lipoma

- ◆ Beneath synovial membrane of the joint
- ◆ Rounded or oval swelling related to a joint with normal skin overlying.
- ◆ If around the knee it can be differentiated from baker's cyst by being constant in consistency whether the joint is flexed or extended.

4. Submucous lipoma

- ◆ Beneath mucous membrane of larynx → it may cause respiratory obstruction.
- ◆ Beneath mucous membrane of the intestine → it may cause intussusception.



Endoscopic view of submucous lipoma of the intestine

5. Inter or intramuscular

- ◆ Swelling in or in between muscles, common in thigh, shoulder with normal skin overlying.
- ◆ It becomes more firm on muscle contraction.
- ◆ **DD:** fibrosarcoma (hard & rapidly growing)

6. Subperiosteal lipoma

- ◆ Swelling in relation to long or flat bones.
- ◆ It is difficult to be differentiated from osteoma.

7. Extradural

- ◆ Only in spinal cord, **never in the cranium as there is no fat.**
- ◆ Presented by pressure symptoms.

8. Subserous lipoma

- ◆ Beneath visceral or parietal peritoneum or pleura.

9. Intraglandular lipoma

- ◆ Inside the gland as parotid gland, thyroid gland and breast

10. Retroperitoneal lipoma

- ◆ Behind post. parietal peritoneum.

11. Lipoma in the suprasternal area (Burn's space)

(rare type)

Diffuse lipomatosis "Dercum disease":

1. Type of patient: Female, usually post menopausal.
2. Site: Usually in lower limb.
3. Characterized by small, multiple swellings and painful.

Complications**1. Dangerous types of lipoma:**

- Submucous lipoma: as it may cause respiratory obstruction or intussusceptions.
- Retroperitoneal: as it turn malignant.
- Some surgeons believe that liposarcoma is malignant from the start.
- Extradural lipoma: as it causes pressure manifestations in spinal cord only

2. Infection.**3. Ulceration** of overlying skin and mucous membranes.**4. Degenerative changes**, liquefaction or calcification.**Investigations****- Specific:**

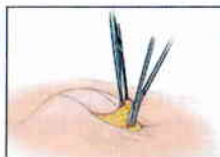
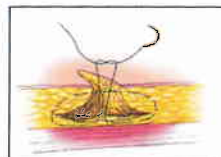
- Excisional biopsy.
- X-Ray: if Subperiosteal.
- CT spinal cord if it is extradural.

DD

- According to its site e.g. Subperiosteal should be differentiated from osteoma.

Treatment

- Surgical excision (Enucleation of tumor from its capsule) if:
 - Not accepted cosmetically.
 - Complicated.

*Incision**Encularion**Suturing***Prognosis**

- Subcutaneous lipoma never to turn malignant, but in thighs and buttocks there is increased incidence of malignant transformation.
- Retroperitoneal lipoma: more liable to turn malignant.

Oral Questions

- Dercum disease
- Dangerous types of lipoma
- Painful lipoma (neuropiloma, Dercum, Complicated lipoma)

Tumors of the Nerves**1- Neurofibromatosis****2- Ganglioneuroma:**

- It is a benign tumor, arises from the ganglion of sympathetic trunk or the suprarenal gland (medulla) and usually affects children
- Usually presents as round swelling in the abdomen or space occupying lesion in the chest
- Treatment: Excision (well encapsulated thus It is easily enucleated)

3- Neuroblastoma: Differential diagnosis (Wilm's tumor)

- it is a highly malignant tumor, affects young infants
- It arises from the sympathetic chain or suprarenal medulla
- The patients presents with a rapidly growing swelling, reddish purple, soft +/- secondaries (blood and lymph) especially for bone marrow

4- Stump neuroma:

- It is a traumatic neuroma, arising in a divided nerve
- It is composed of fibrous tissue, growing axons and neurolemma cells
- **Types:**
 - Painless or painful
 - Terminal, central or lateral
- **Treatment:** Excision +/- sympathectomy in cases with neuralgia

Neurofibroma

Introduction

- neuron is formed of (cell body, nerve axon, dendritis) → it is the functional unit of nervous system
- Nerve axon is surrounded by Schwann cell which produce neurolemmal sheath

- **Types:** commonest is generalized
- **C/P:** swelling + café au lait patches
- **Comp.:** pressure, neurofibrosarcoma
- **Invest.:** only in familial type
- **TTT:** excision

Definition

- It is a tumor like masses formed from nerve sheaths either neurolemma (neurolemmoma) of spinal nerves or schwannoma from cranial nerves usually the 8th nerve.
- It is considered as hamartoma.

Types

- 1- Generalized (Von Reckling Hausen's disease)
- 2- Solitary.
- 3- Patchy dermatocele (Plexiform neuroma).
- 4- Molluscum Fibrosum.
- 5- Elephantiasis neurofibromatosis.
- 6- Acoustic (8th nerve)

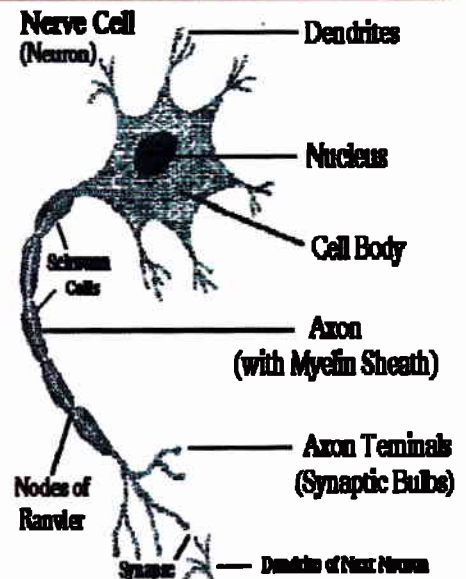
Clinical Picture

Symptoms

- Patient usually presented with painless swelling that moves across and not along the nerves of gradual onset and slowly progressive course.

O/E

- **No. :** Solitary or multiple.
- **Size:** variable.
- **Shape:** rounded.
- **Site:** subcutaneous.
- **Edge:** well defined.
- **Consistency:** firm.
- **Mobility:** not attached to skin.
- **Café au lait patches:** brown patches seen all over the body.



→ ACCORDING TO DIFFERENT TYPES:

1- Generalized neurofibromatosis (Von Recklinghausen's disease)

- The most common type
 - Often it is familial
 - No. : multiple
 - Size: variable
 - Consistency: firm.
 - Mobility: mobile across but not along the nerve.
 - Café au lait patches: especially on the back.
 - Special characters: tender, ↑ ICT if arise from the intracranial nerve, no sensory or motor loss.
 - Pheochromocytoma may be present (MEN IIb).
 - Malignant transformation is associated with pain, anesthesia, paralysis and progressive increase in the size of lesion.

2- Solitary neurofibroma

- No.: single.
- Size: variable.
- Consistency: firm, tender (due to compression of nearby nerve fibers-not infiltration).
- Mobility: across but not along the nerve
- Café au lait patches.

3- Pachydermatocele (Plexiform Neuroma)

- Cystic swelling gives sensation as a bag of worms due to presence of multiple nerve fibers inside the swelling
- swelling is common in face → facial deformity

4- Molluscum fibrosum

- Soft fibrous swellings (cutaneous nerve endings are affected)
- Scalp, face and trunk are commonly affected (hand and feet escape).

5- Elephantiasis neurofibromatosis:

- Skin becomes thickened and replaced by grayish white glistening tissue (diffuse affection of the nerves of the skin and subcutaneous tissue).
- Commonly in children in limbs.
- Hypertrophy of a huge part of a limb.
- Differentiated from lymphedema by congenital nature & café au lait patches.

6- Acoustic neuroma :

- May be associated with acoustic nerve tumor.
- It has a striking familial incidence showing itself as Mendelian dominant trait.
- It is often associated with tumors of the dura and choroid plexus.

Neurofibrosarcoma :

- Arise de novo or may develop by malignant transformation in a neurofibroma.
- It forms a painful rapidly growing tumor which invades the surrounding tissues producing anesthesia and paralysis and forms distant metastasis.
- Wide excision should be carried out in localized tumors, but amputation is indicated for extended tumors and post operative recurrence.

Complications

- 1- Pressure symptoms e.g. deafness in acoustic neuroma.
- 2- malignant transformation(neurofibrosarcoma):
 - Sudden change in behaviour :-pain -rapid growth-hard in consistency-never paralysis.

D.D. (Multiple swellings)

1. Multiple neurofibromatosis.
2. Multiple lipomatosis.
3. Multiple melanoma
4. Multiple angioma.
5. Multiple lymph node enlargement.
6. Multiple warts.
7. Multiple exostosis (bony swellings).

Investigations Mainly clinical diagnosis

- Search for associated conditions e.g. MENIIb (blood pressure-serum calcitonin-urinary catecholamines).

Treatment

- Surgical excision is the treatment of choice
- **BUT**
 1. Debulking of the lesion is necessary to improve self image of the patient.
 2. With high rate of recurrence till age of puberty → repeated partial excision.
 3. Patient with generalized neurofibromatosis → must be fully examined because they might have acoustic neuroma.

▪ **Neurofibroma does not affect nerves except neurofibrosarcoma and acoustic neuroma.**

▪ **Familial neurofibroma: Von Reckling Hausen disease and MENIIb.**

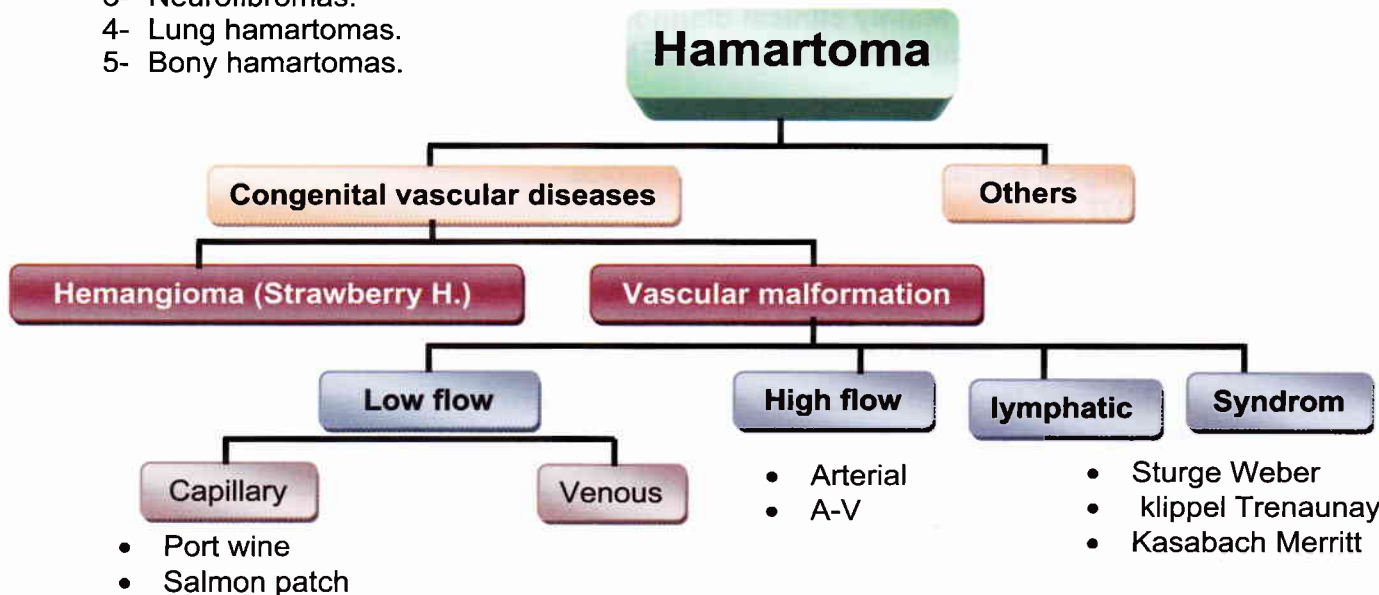
Hamartoma

Definition

- Mal-arrangement of normal tissue.

Examples include:

- 1- Congenital vascular diseases
 - a. Hemangioma
 - b. Vascular malformations
- 2- Pigmented skin lesions.
 - a. Naveus
 - b. Melanocyte tumors
- 3- Neurofibromas.
- 4- Lung hamartomas.
- 5- Bony hamartomas.



I- Congenital Vascular Diseases

A-Hemangioma (strawberry hemangioma)

Definition

- Benign tumor of endothelial cells of capillaries.

Incidence

- Most common & most rapidly growing tumor in infancy.
- 70% are present at birth and 30% appear within the 1st year of life.
- Female : Male = 3 : 1
- 10% of white infants.

- **Incid.:** commonest benign tumor in infancy, (♀: ♂=3:1)
- **C/P:** patch ↑, →, ↓ in size
- **Comp.:** site dependant
- **TTT:** mainly reassurance

Pathology

- **Site:** Anybody surface but the most common site is face.
- **The lesion usually presents by the following 3 stages:**
 1. **Stage of proliferation** short after birth a pale or reddish patch and increases in size over 6 months.
 2. **Involution phase:** usually starts at the end of first year , central pallor appears and the color of the lesion slowly fades. There is progressive decrease in the size of the lesion. This stage continues till the age of 10 years.
 3. **Involuted phase:** The skin finally is normal in 50% of patients. Some residual skin changes as discoloration, scarring, telangiectasia may present.

Clinical picture

- Lesion start as erythematous raised patch with irregular surface
- During the 1st 2-3 wks after birth
- grows stationary for 2-3 yrs
- Then spontaneous regression starts & completed by age of 5-7 yrs

Complication

According to the site

1. In the face → Squint.
2. In the eye lid → ↓ visual field → amblyopia + blindness.
3. Ulceration is a common complication.
4. Bleeding is not common.
5. Infection .
6. Airway obstruction.
7. Large visceral haemangioma may entrap platelets leading to thrombocytopenia.
8. Large visceral or cutaneous haemangioma may lead to congestive heart failure.

Treatment

1. Reassurance of the parents and observation are recommended as the appearance of skin after involution may be better than the scar following incision
2. **When it is necessary to interfere the following are helpful:**
 - a. Oral or intralesional steroids.
 - b. Laser photo coagulation.
 - c. Surgical debulking

B. Vascular malformations

Definition

- **Malformation of vascular development in utero.**

Incidence

- **Male = female**

Types

- I. **Low flow malformation**
 1. Capillary
 - Portwine stain (nevus flavus).
 - Salmon pink patch
 2. Venous = cavernous
- II. **High flow malformation**
 1. Arterial malformation
 2. A-V malformation
- III. **Lymphatic.**

Pathology

They are vascular malformation that resemble tissue of origin (arterial , venous , capillary & lymphatic)

So:

- They always present at birth
- Shows no spontaneous involution

Diagnostic imaging

- 1- **Doppler U/S** → to differentiate high flow & low flow.
- 2- **MRI or MRA** → to assess the extent & involvement of the structure.
- 3- **Angiography** in some cases.

I- Low flow Malformations

A. Capillary malformations

I- Portwine stain

- Commonest type of vascular malformation
- The lesion is present since birth and it doesn't undergo involution.
- The lesion is dark purple in color and it doesn't raise above the surface. Pressure causes blanching but the color returns immediately after release of pressure.
- Sometimes the lesion takes the distribution of one of the branches of trigeminal nerve , but the lesion doesn't cross midline.
- Sometimes a portwine stain of the face is associated with similar lesions in the meninges (**Sturge Weber Syndrome**).



➔ Treatment :

- a. Pulsed dye laser is the treatment of choice during childhood. Requires general anaesthesia and multiple sessions may be needed.
- b. YAG laser is the treatment during adulthood. Results are not as favorable as children.

II- Salmon Patch

- Nearly half of all babies have salmon patch.
- Appear as pink, flat marks on the forehead, back of the neck or on the lip.
- The skin is not thickened.
- Usually covered by hair and out of sight (at the back of neck).



B. Venous Malformations

(Previously called cavernous hemangioma)

Definition

- Multiple large inter communicating sinus (vascular spaces)

Incidence

- less common than capillary (portwine)

Site

- External: Cheeks, lips Tongue, eye, ear extremitiesect
- Internal: the commonest site is liver.

Clinical picture

- 1- Present since birth / no spontaneous involution.
- 2- Compressible swelling with some discoloration of overlying skin.
- 3- Sign of emptying is very characteristic: Pressure causes blenching but colour returns immediately after release of pressure.
- 4- If in the liver → sequestration of platelets → thrombocytopenic purpra
- 5- Add clinical picture of the site (tongue, lips , earect)

Investigation

- 1- Arteriography: pre-operative : shows extent of the lesion (both diagnostic & therapeutic).
- 2- CT Scan : to show extent of the lesion

Treatment

- 1- Compression therapy may relieve pain and edema.
- 2- Percutaneous sclerosis may be performed with hypertonic saline or 100% alcohol. There is a high recurrence rate; multiple sessions may be required.
- 3- Surgical excision. Preoperative embolization or sclerosis facilitates intraoperative haemostasis.
- 4- Interstitial laser coagulation.

II- High flow malformations

I- Arterial (plexiform hemangioma) (Cersoid aneurysm)

Definition

- It is congenital A-V fistula.

Pathology

Site

- Usually in the scalp, especially in temporal region or occipital region may involve underlying bone (in relation to superficial temporal artery).

- **C/P:** swelling (disfigurement), headache, comp.
- **Comp.:** as cyst
- **Invest.:** Doppler, angiography
- **TTT:** excision

Macroscopic picture

- A bluish cystic swelling

Microscopic picture

- Consists of arteries and veins

Diagnosis

- Clinical picture

- **Complaint:**

- ♦ Cosmetic disfigurement.
- ♦ Headache.

- **Signs:**

- ♦ **General:**

- Fever if inflamed
- Water hammer pulse.

- ♦ **Local:**

- Inspection:
 - Irregular swelling at temporal region, with normal skin overlying.
- Palpation: pulsating compressible swelling.
- Auscultation: machinery murmur over it.

Complications

- Hemorrhage (very dangerous as it's arterial).
- Cosmetic disfigurement.
- Infection.
- Ulceration of skin overlying.
- Degenerative changes e.g. calcification.

DD

- From other types of Hemangioma e.g. cavernous Hemangioma.

Investigations

- Doppler and duplex.
- External carotid angiography.
- X-Ray on the skull → shows rarefaction of the bone.

Treatment

Surgical TTT is very difficult d.t. presence of multiple feeding arteries

- Surgical excision with the following precautions:
 - Semisitting position.
 - General hypotensive anesthesia.
 - Preoperative embolization.
 - Temporary ligation of the external carotid artery.

II- Arterio-Venous Malformation

- Characterized by abnormal connections between arteries and veins without an intervening capillary bed.
- Present at birth but may not become evident until late childhood.
- It may be localized or diffuse.
- Localized AVM presents by a localized swelling with increase surface temperature. There may be palpable thrill or murmur.
- Generalized AVM growth disturbance or skeletal distortion.

➔ Treatment :

- a. Ligation or embolization feeding vessel often results in rapid enlargement of collateral vessels increasing the size of the lesion.
- b. Excision .Preoperative Angiography and embolization of feeding arteries followed by excision next day. Extensive coverage by a free flap may be needed.

■ Hereditary hemorrhagic telangiectasia

- Is an autosomal dominant disease characterized by multiple cutaneous, visceral an mucosal AVM

Hemangioma	Vascular malformation
▪ Appears shortly after birth. ▪ 3 stages: – Progressive. – Stationary. – Regressive.	▪ Dated since birth. ▪ Stationary or slowly progressive.

III. Lymphatic Malformation

- Classified as microcystic or macrocystic and may have a component of venous malformation.
- The head & neck or the axilla are affected in majority of cases. Neck masses may extend into the mediastinum.
- LMs are the most common cause of congenital macroglossia , macrocheilia & macro-otia.
- Skeletal involvement may cause distortion

➔ Treatment :

- a. Percutaneous aspiration followed by sclerosis may provide short term improvement; usually requires additional treatment.
- b. Surgical excision often requires multiple staged procedures. Usually wait until the age of 3 years.

IV. Other Vascular Malformation

1. Glomus Tumor (Angioneuroma)

- **Definition:** The tumor arising from A-V shunt in extremities
- **Pathology:** Vascular, neuron & smooth muscle fibers elements.
- **Clinical picture :**
 - Appear as minute very tender purple spot under the nail
 - Paroxysmal pain is induced by pressure or change in temperature
- **Treatment:** by excision

2. Spider nevi

- Lesion arise in patient with liver cirrhosis
- May be due to hyperestrogenism
- Lesion consist of small tiny arterial nevus which radiate multiple capillaries.

3. Hereditary hemorrhagic telangiectasia

- They are multiple angiomas in the skin & mucosa with strong tendency to bleed

Older classification

I. Descriptive classification

- 1- Port wine stain
- 2- Salmon patch
- 3- Strawberry (cavernous) hemangioma
- 4- Cirsoid aneurysm

II. Based on cell origin

- 1- Capillary malformation
- 2- Venous malformation
- 3- Arterial malformation
- 4- Lymphatic malformation

III. Recent (based on endothelial cell characters)

- 1- Hemangioma
- 2- vascular malformation

More details

Sturge Weber Syndrome

1. Lepto-meningial A-V malformation covering comprises over sensory & motor area
2. Capillary vascular malformation in face not crossing midline
3. commonly associated with portwine stain
4. A-V malformation in extremities (gigantism seen in middle finger, left thumb, scrotum, whole lower limb)

Klippel Trenaunay Syndrome

- Port wine + 2ry various veins due to A-V malformation

Cysts in Surgery

Definition

- A collection of fluid in a sac.

Classification

▪ According to lining endothelium:

True cyst	False cyst
□ Lined by endothelium or epithelium which is usually responsible for the secretion of fluid	□ Result from discharge or degeneration.
□ Thyroglossal cyst □ Dermoid cyst. □ Bartholin's cyst.	□ Pseudocyst of pancreas □ Cystic degeneration in a tumor

▪ According to etiology:

A. Congenital:

1. Sequestration dermoid:
 - Due to:
 - Displacement of epithelium along the suture line during closure.
2. Tubulodermoid / Tubuloepidermoid:
 - Due to:
 - Abnormal budding e.g. thyroglossal cyst.
 - Dilation of vestigial remnants e.g. urachal cyst, vitello-intestinal cysts, Hydatid cyst of Morgagni and branchial cysts.
3. Cysts of embryonic origin:

B. Acquired:

1. Retention cysts:
 - Due to:
 - Blocking of a glandular duct e.g. sebaceous cyst, ranula, bartholin's gland cyst or hydronephrosis.
2. Distention cysts: e.g. dilated acini of thyroid follicles or in the ovary or lymphatic cyst as cystic hygroma.
3. Cystic tumors e.g. cystadenoma, cystadenocarcinoma of the ovary.
4. Parasitic cysts e.g. hydatid cysts.
5. Pseudocysts:
 - Due to:
 - Liquefaction by necrosis or hemorrhage e.g. necrosis tumors.
6. Exudation cyst: exudation into an anatomical space already lined with epithelium e.g. hydrocele.
7. Traumatic cysts: hematoma may resolve into a cyst which becomes lined with endothelium (cure requires excision not aspiration).

Sebacaceous Cyst (Epidermoid Cyst)

Definition

- Retention cyst of sebaceous gland.

Etiology

- **Acquired cyst caused by:**
 - Obstruction of sebaceous gland duct by inspissated sebum-Drills- scarring.
 - Result in: Retention of sebaceous secretion into sebaceous gland.

- **C/P:** painless swelling, punctum, sebum
- **Comp.:** cyst + horn + cock's peculiar t.
- **D.D.:** dermoid cyst
- **Invest.:** C&S + FBS in recurrent infection
- **TTT:** excision + ttt of comp.

Pathology

Sites

- Anywhere in the skin related to hair (scalp, axilla & groin).
- *Never in palms and sole "No sebaceous glands"*

Macroscopic

- Cystic swelling attached to skin at a point "**Punctum**"
- Contains: semisolid, greasy sebaceous, material with bad odour.

Microscopic

- A true cyst: lined by flat squamous epithelium.

Diagnosis

Clinical picture:

- **Age:** (rarely before adolescence)
- **Complaint:** a slowly growing painless subcutaneous swelling.
- **Signs:**
 - It is well defined, small, tense, cystic swelling, indentable, non-compressible (to differentiate between it & hemangioma).
 - Attached to the skin at one point which is the site of the duct (punctum) "**black spot**"
 - On squeezing it discharges sebum.
 - It is slowly growing, may attain large size and may be solitary or multiple.

NB. If inflamed → ↑size, tender, fixed to deep structures

Complications

As any cyst

- 1- Commonest → infection and suppuration
- 2- Sebaceous horn → dried → inspissated sebum. Protrude from the punctum over the skin.
- 3- Cock's peculiar tumor (very rare) → (ulceration of sebaceous horn)
 - **NB. Simulate squamous cell carcinoma but base is not indurated**
- 4- Local alopecia: due to stretch of the skin for long time.



Cock's peculiar tumor



Sebaceous horn in ear

DD

- 1- Dermoid cyst 2- Cock's peculiar tumor (SCC. BSC)

	Sebaceous cyst	Dermoid cyst
Age	After adolescence	At childhood
Site	Related to hairy skin	Related to lines of fusion
Consistency	Tense cystic	Lax cystic
Skin	Attached to skin by punctum where sebum can be squeezed	Not attached to skin

Investigations

- Not important. It is a clinical diagnosis.
- May be needed with complications:
 - 1- Biopsy: in cock's peculiar tumor.
 - 2- Culture and sensitivity with recurrent infection.
 - 3- Fasting blood sugar with recurrence of infection in spite of adequate treatment

Treatment

- 1- Uncomplicated → excision through an elliptical incision to include the punctum.
- 2- Infected → Incision and drainage
→ After inflammation subsides → excision

Multiple cysts of the scrotum → excision of the skin bearing the cysts

Dermoid Cyst

Types

- 1- Sequestration dermoid cyst.
- 2- Implantation dermoid cyst.
 - Epidermoid cyst.
- 3- Tubulodermoid cyst:
 - Thyroglossal cyst.
 - Branchial cyst.
- 4- Teratomatous dermoid cyst of the ovary and testis (sacroccocygeal tumor).
- 5- Inclusion dermoid.

Sequestration Dermoid Cyst

Definition

- A congenital true cyst lined by squamous epithelium.

- **Etiology:** cong. true cyst
- **C/P:** painless swelling (lax, not attached to skin)
- **Comp.:** cyst + intracranial comp.
- **Invest.:** skull x-ray
- **TTT:** excision + ttt of comp.

Etiology

- A congenital cyst.
- Caused by sequestered piece of epithelium in the subcutaneous tissue at a line of fusion of the body during fetal life but it appears later in life.



External angular dermoid cyst

Pathology

Site

▪ Along the lines of fusion:

- 1- Face → external angular (commonest)
→ Internal angular
- 2- Ear → post-auricular
→ Pre-auricular
- 3- Neck → in midline
→ Sublingual or supramyelohyoid (in the floor of the mouth)
→ Inframylahyoid.
→ suprasternal
- 4- Trunk → in midline

Never in a limb (develop from buds → no line of fusion)

Macroscopic

- Cystic swelling.
- Contains: yellowish greasy sebaceous material.

Microscopic

- Outer fibrous layers.
- Wall lined by stratified squamous epithelium.

Diagnosis

Clinical Picture

- Age: at childhood
- Complaint:
 - A slowly growing painless subcutaneous swelling related to the previously mentioned sites.
- Signs:
 - ⇒ General:
 - Other anomalies.
 - ⇒ Local
 - It presents as well defined, globular, **lax**, cystic swelling which is **not attached to the skin**.
 - The underlying bone may be hollowed out.
 - There may be a **pedicle connecting the deep aspect of the cyst to dura matter**.

External angular dermoid cysts lie on bone defect caused by constant pressure

Complications

As any cyst (Infection, rupture, hemorrhage, pressure)

- 1- Cerebral compression (very rare) → Dumble shaped or hourglass dermoid
- 2- Intracranial complication with the dura matter should be investigated by skull x-ray before excision to be sure the sutures are closed.
- 3- Recurrence after excision → if incompletely excised

DD

From sebaceous cyst

Investigations

- Skull x-ray → to exclude presence of bone defects (if present, we wait till it closes)

Treatment

- 1- Uncomplicated
 - Surgical excision (best lines of treatment)
 - In children with dermoid cyst in the scalp it is better to wait till closure of the skull sutures because some cysts may communicate with the dura.
- 2- Infected → incision and drainage followed by excision after inflammation subsides.

Implantation Dermoid Cyst

Definition

- Acquired dermoid cyst

Etiology

- Implantation of skin into subcutaneous tissue by:
 - 1- Pricking wound on the tip of a finger as during sewing.
 - 2- Use of skin as graft in hernioplasty.

Pathology

Site

- In a tip of finger.
- Related to scar traumatic or surgical.

Macroscopic & Microscopic

- See before

Diagnosis

Type of Patient

- Sewer or manual worker with a swelling related to tip of finger

Complaint

- Slowly growing swelling at 1st painless then painful

Signs

- They are small & tense cystic swellings.
- The overlying skin is sometimes scarred.
- It may be tender due to compression on nerve endings at pulp of fingers.



Complications and Treatment

- See before.

Inclusion dermoids

- These are due to inclusion of the epidermis during closure of a cavity.
- They include sublingual, suprasternal, intracranial and intraspinal dermoids

Teratomatous dermoids

- These are benign forms of teratomas.
- The cyst is lined by squamous epithelium but contains teeth, hair, bone or cartilage.
- They occur most commonly in the ovary but may be found in the testis or posterior mediastinum.

Thyroglossal Cyst

Embryology

- Thyroglossal trunk develops from the foramen caecum of the tongue and descends into the neck → gives thyroid isthmus and medial part of thyroid lobes.

Etiology

- From unobliterated portion of thyroglossal duct (which develop from foramen caecum and descend into the neck to give thyroid isthmus and medial parts of the lobes).
- Site:**
 - At any point of the course of thyroglossal tract.
 - The commonest site is "subhyoid in midline" then on (thyroid cartilage usually pushed to left side) and rarely "suprahyoid"

Pathology

- Wall of cyst:** may contain thyroid tissue.
- Lined with:** stratified squamous epithelium.
- Containing:** sebaceous material.
- Fibrous cord:** connects the cyst to hyoid bone (it can be followed to foramen caecum at the base of the tongue).



Clinical Picture

- Type of patient:** child 6-8 years presented by swelling in the front of the neck.
- Symptoms:**
 - Painless mass in the midline of the neck.
 - Pain if infected (the commonest complication as it is rich in lymphatics)
- Signs:**
 - Cystic mass in midline of the neck (by Paget's test).
 - Mobility:
 - Moves with deglutition and protrusion of the tongue (**due to attachment to foramen caecum of the tongue**)
 - Moves from side to side not from above downwards.
 - There may be a palpable track extending from hyoid bone upwards towards tongue.



Complications

- As any cyst (infection, rupture, hemorrhage, pressure, calcification)
- Thyroglossal fistula:
 - Etiology:** Acquired fistula (**never congenital**) due to:
 - Infection of thyroglossal cyst leading to rupture.
 - Inadequate excision of the cyst.

C/P:

- Discharges viscous fluid or pus.
- The opening is near midline or to the Lt. side crescentic due to fibrosis.



Infected thyroglossal cyst

DD

- Ectopic thyroid.

Investigations

- U/S → cyst.
- Fistulogram → if fistula coexists.
- Routine preoperative investigations.



Thyroglossal fistula

Treatment

- **Sistrunk operation:**
 - **Excision of:**
 - Cyst.
 - Central part of hyoid bone (to avoid recurrence).
 - Core of tissue of the tongue up to foramen caecum.
 - Pyramidal lobe of the thyroid.
 - Track.



After Sistrunk operation

Prognosis

- If incomplete excision → recurrence or fistula may result.

Branchial Cyst

Embryology

- Normally the 2nd branchial arch grows rapidly covering the 3rd and 4th arches then fuse with the 5th arch
- Resulting in a space called (cervical sinus).

- **Etiology:** persistent cervical sinus
- **C/P:** painless swelling + comp.
- **Comp.:** cyst, fistula(cong.), cancer
- **Invest.:** U/S ± fistulogram
- **TTT:** total surgical excision
- stepladder op. for fistula

Etiology

- Cervical sinus persists → branchial cyst.
- ± 2nd arch does not completely fuses with the 5th arch → congenital branchial fistula.

Pathology

Site

- Upper part of the neck.
- Related to carotid triangle.
- Partially deep to sternomastoid muscle protruding beneath its anterior border.

Macroscopic

- A cyst with a fibrous cord passing between external and internal carotids
- Connect cyst to pharynx "end above tonsil".

The track extend up to side wall of nasopharynx "fossa of Rosenmuller"

Microscopic

- Lined by squamous epithelium.
- Contain mucoid substance rich in cholesterol (rectangular crystals with one missing angle)
- Rich in lymphoid tissue so it is liable to infection.

DD

- **Cysts rich in cholesterol:**
 - Branchial cyst.
 - Spermatocoele.
 - Dental-dentigerous cyst.
 - Primary vaginal hydrocele.

Clinical Picture

Age

- Although congenital, it appears at the age of 20 years



Complaint

- a slowly growing painless swelling at lateral side of the upper part of the neck

Signs

General:

- FAHM if infected swelling developed.

Local:

- **Site:** protrude beneath the anterior border of upper 1/3 of sternomastoid muscle
- **Shape:** rounded or oval
- **Surface:** smooth
- **Edge:** well defined
- **Consistency:** tense
- **May be pulsating , compressible**
- **Skin overlying:** normal
- **Special signs:** on it bulges out.

Complications

As any cyst

- 1- Infection: is the commonest
- 2- Acquired branchial fistula due to rupture, incomplete excision or incomplete incision of the abscess,
 - Fistula appears at anterior border of lower third of sternomastoid muscle it is crescentic in shape, discharging mucoid material or pus if infected.
- 3- Adenocarcinoma: very rare ☑

Branchial fistula may be

Congenital (Usually)

- 1- Since birth
- 2- Longer.
- 3- No scar

Acquired (Rare)

- 1- Later on in life
- 2- shorter
- 3- Related to scar.

DD

Swelling at lateral side of the neck

- 1- Cold abscess (TB C/P)
- 2- Cyst in a thyroid lobe.
- 3- Laryngocele, pneumatocele.
- 4- Carotid artery aneurysm.
- 5- Fistula with TB sinus.



Branchial fistula

Investigations

➤ **If fistula** → fistulogram.

➤ **If cyst:**

U/S:

- Ensure the diagnosis.
- Exclude DD as cold abscess.

▪ **Discharge** → Ziehl Neelsen stain

- Characteristic rectangular cholesterol crystals with missing angles.

▪ **Routine preoperative.**

DD

1. Branchial cyst
2. Cold abscess
3. Cyst in thyroid lobe
4. Laryngocele & pneumatocele
5. Carotid artery aneurysm

Treatment

- **Cyst:**
 - Complete excision through transverse neck incision.
- **Fistula:**
 - Excision through multiple transverse neck incisions to remove the whole tract (Step ladder operation).
 - Ureteric catheter may be introduced in the track to facilitate the operation.

Ganglion**Types**

1. Simple Ganglion.
2. Compound palmar ganglion.
3. Giant tumor of tendon sheath.
4. Glomus tumor.
5. Implantation dermoid cyst.

- **Etiology:** mucoid degeneration
- **C/P:** painless cystic swelling
- **Comp.:** cyst + cancer
- **Invest.:** x-ray, excisional biopsy
- **TTT:** excision

1.Simple Ganglion

➤ 95% of hand swellings are benign

- **Definition**
 - Chronic cyst containing mucoid material related to a tendon.
- **Etiology**
 - Mucoid degeneration of fibrous tissue of tendon sheath.
- **Pathology**
 - Cyst contains jelly like mucin.
- **Clinical Picture**
 - ⇒ **Symptoms**
 - Painless swelling at dorsum of the hand or around the ankle.
 - ⇒ **Signs**
 - **General:** may be fever if infection.
 - **Local:** localized, tense, **cystic** (by Paget test), rounded swelling, moves across the tendon. its mobility is ↓ by pulling on the tendon.
- **Complications**
 - 1- Infection.
 - 2- Hemorrhage.
 - 3- Calcification.
 - 4- Rupture.
 - 5- Malignant changes.
- **Investigations**
 - X-ray on hand.
 - Excisional biopsy.
- **Treatment**
 - It is not indicated unless the patient insists.
 - Aspiration or rupture of cyst by applying direct pressure
 - Complete excision under GA until its root in the presence of good light, tourniquet & bloodless field.



2. Compound Palmar Ganglion (TB of ulnar bursa)

- **Definition:**
 - TB synovitis of synovial flexor sheath at the fingers → distended with TB granulation tissues.
- **Etiology:**
 - Causative organism: Mycobacterium TB.
 - Route of infection:
 - Direct, inoculation.
 - Lymphatic spread.
 - Blood spread.
- **Pathology:**
 - ⇒ **Site:** - Palm. - Distal part of the forearm.
 - ⇒ **Macroscopic:**
 - Aggregation of TB follicles lymphangitis and lymphadenitis.
 - ⇒ **Microscopic:**
 - TB tubercle formed of central caseation surrounded by epithelial cells, giant langhans cells fibroblasts and fibrocysts.
- **Clinical Picture:**
 - ⇒ **Symptoms:**
 - Swelling at palm of the hand and distal part of the forearm.
 - May be on associated TB toxemia, night fever, night sweating, anorexia and loss of weight.
 - ⇒ **Signs:**
 - General: TB toxemia.
 - Local:
 - Inspection:
 - Swelling at palm and distal part of forearm, larger in size, may extend to thumb or little finger.
 - Overlying skin may show signs of inflammation.
 - Palpation:
 - Swelling with smooth surface, ill-defined edge, cystic in consistency and fluctuant: show "**cross-fluctuation**" due to band of construction at flexor retinaculum.
 - LNs may show TB lymphadenitis.
- **Complications:**
 - 1- Secondary infection. 2- TB spread and toxemia.
 - 3- Hemorrhage. 4- Rupture.
- **Investigations:**
 - ⇒ **Lab:**
 - Tuberculin test. - CBC → leukopenia with relative lymphocytosis.
 - ESR >100
 - Aspiration and culture on Lowenstein Jensen media.
 - PCR.
 - ⇒ **Imaging:** CXR.
- **Treatment:**
 - ⇒ **Medical treatment:**
 - Sanatorial treatment. - Antituberculous drugs.
 - Immobilization in plaster of paris.
 - ⇒ **Surgical:**
 - Aspiration by z-technique.
 - If failed → Complete excision under umbrella of anti-tuberculous drugs.

▪ **Prognosis:**

- Depends on a patient's resistance and virulence, and load of bacteria.
- Good immunity → fibrosis.
- Low immunity.
- Coalesce.
- Caseation.
- Cold abscess.

3. Giant cell tumor of tendon sheath:

- Painless mass on volar or dorsal aspects of finger.
- Interfere with tendon movements, compress digital nerve & erode bone.
- **Treatment:**
 - Metictular excision.
 - Recurrence is treated by re-excision combined with radiotherapy.

4. Glomus tumor: discussed before.

5. Implantation dermoid cyst: discussed before.

Cystic Hygroma (Cavernous Lymphangioma)

Definition

- Dilated cavernous lymph spaces

Etiology

- Diffuse lymphangioma it is a hamartoma rather than true tumor of lymphatic vessels.
- It is due to failure of sequestration of part of jugular lymph sac of fetus.

- **Etiology:** hamartoma
- **C/P:** lax cystic **translucent** partially compressible non-pulsating swelling
- **Comp.:** inf., resp. distress, obst. labour
- **TTT:** excision

Pathology

Site

- **Commonest sites** → root of the neck (superficial to sternomastoid M. & extends to posterior triangle)
- **Less common in** → lip (macrocheilia)
→ tongue (macroglossia)
→ axilla
→ mediastinum
→ groin

Microscopic

- Multiple intercommunicated lymph spaces lined by endothelial cells and contain lymph. Cysts near surface are large while deeper one are small and infiltrate muscle.

Macroscopic

- Size → large
- Shape → irregular
- Edge → ill defined
- Color → translucent

Clinical Picture

Type of patient:

- At birth or within first few years of life

Swelling

- Number → single
- Site → lower part of posterior triangle
- Size → large



- Consistency → lax, cystic
- Surface → irregular
- Edge → ill defined
- Transillumination → **Translucent** (the only translucent neck swelling)
- Special character → partially compressible, non-pulsating mass.
- Increase in size on coughing or crying.

DD

- Branchial cyst (deep to sternomastoid & anterior)

Complication

1. Obstructed labour.
2. Recurrent infection (rich in lymphatic).
3. Respiratory distress due to compression of trachea to induce fibrosis.

Treatment

- Surgical excision of the swelling at about the age of 3 years should be done. This could be facilitated by injection of boiling water in the swelling to introduce fibrosis to make it smaller

Complication of surgery: injury to facial N.

Laryngocele

Definition

- Herniation of the mucous membrane through a weak point in the thyrohyoid membrane

Clinical Picture

- Cystic swelling in the neck
- Which becomes prominent on straining
- Occurs in musicians
- Swelling is resonant and compressible.

**Treatment**

- Factor of straining should be corrected
- Excision

Pneumatocele

Definition

- Herniation of the lung apex through weak **Sibson's fascia** (suprapleural membrane) which extends from the transverse process of C₇ to the 1st rib.

Clinical Picture

- Cystic swelling in the supracalvicular region.
- Becomes prominent on straining.
- Resonant & compressible

Treatment

- Factor of straining should be corrected.
- Plication of Sibson's fascia

Subhyoid bursitis

- This is a rare tender oval swelling which lies transversely below the hyoid bone.
- It moves up & down with deglutition and with protrusion of the tongue.

Cysts of the Mouth Floor

[1] Sublingual Dermoid Cyst

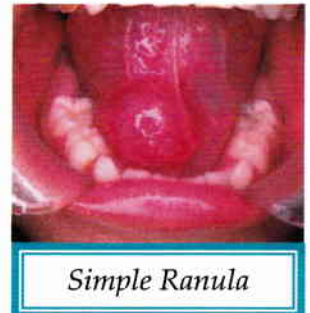
- See skin surgery

[2] Mucous Cyst

- **Definition:** Mucous retention cyst of sublingual salivary gland (Nuhn & Blandin).
- **Site:**
 - On one side of the midline (**DD: dermoid cyst in the midline**).
 - May extend into the neck (behind myelohyoid M.) called plunging ranula of the tongue.

[3] Ranula

- It is a retention cyst arising from sublingual salivary gland.
- Pathology:
 - The wall is composed of: thin fibrous capsule, which is lined by macrophages.
 - Contains gelatinous material.
 - If it extended down to the neck over the posterior margin of myelohyoid, it forms "plunging ranula".
 - It may rupture & refill again.
- **C/P:**
 - Cystic, bluish, and translucent swelling with prominent blood vessels over it and stretch submandibular duct.
 - **Overlying:** covered by m.m. & crossed by Wharton's duct.
 - May extend down in the neck → plunging ranula.
- **TTT:**
 - Marsupialization (deroofing) & suture cyst wall to oral mucous membrane
 - Excision (difficult) in recurrent cases.



Simple Ranula

Keloid

Definition

- A dense over growth of granulation tissues after wounds.

Incidence

1. It is especially common in the face, neck, front of chest & abdomen.
2. Negros (common in negros).
3. Tendency to develop keloid is inherited.

Clinical Picture

- The affected scar becomes firm raised above the surface
- Early it is pink or reddish in color (**acute phase**) & later it becomes pale (**chronic phase**).
- Common as postoperative complication of thyroidectomy, so we do collar incision



DD

- Hypertrophied scar (limited to the scar and never grows > months)

Treatment

- Continuous pressure by silicone gel sheets.
- Intralesional steroids.
- Surgically excision. Recurrence rate after simple excision may reach 80%. To minimize recurrence intramarginal excision of the scar is recommended together with intraoperative injection of steroids.

Baker's Cyst

- Herniation of synovial membrane of knee joint through the capsule.
- It simulates semimembranous bursitis, but it occurs in the midline.
- If rupture → severe pain (DD: DVT).



Investigations Doppler US

Treatment Treatment of the cause

Desmoid Tumor (Baget's tumor = fibroma of the rectus sheath)

Definition (*Desmos* = tendon like).

- It is **locally malignant fibrosarcoma** arising from the fibers of anterior rectus sheath. (myofibroblast in musculo-aponeurotic structure).
- Less commonly from posterior rectus sheath or abdominal muscle.

- **Etiology:** FAP or Gardner's s.
- **C/P:** abdominal wall swelling (below & to the Rt. of the umb.)
- **Invest.:** imaging + biopsy
- **TTT:** excision + safety margin

Incidence

- Common in multipara females
- Common with **Gardner's syndrome**.

Etiology

- Part of **FAP** or gardner syndrome
- Previous surgical incision or over stretch of anterior rectus sheath.
- May develop in relation to estrogen levels (↑ by oral contraceptive pills contain estrogen receptors)

Pathology

Macroscopic

- It is hard, pinkish, non-capsulated mass at site of previous surgical incisions.
- Cut section shows barrel fibers like tendon so it is named desmoid tumor.

Microscopic

- It is similar to fibroma but with giant body cells.

Clinical Picture

1) Peripheral desmoid tumors :

- Painless, firm, smooth, and mobile.
- Overlying skin is usually unaffected.
- Often adhere to surrounding structures (early mobile, later fixed).

2) Abdominal wall desmoid tumors :

- It arises from the rectus sheath below level of umbilicus usually to one side (commonly right side) but never in the midline.
- Most commonly arise from the rectus abdominis muscle in postpartum women and in scars due to abdominal surgery
- Slowly growing swelling, firm to hard

3) Intra-abdominal desmoid tumors

- More common in patients with familial polyposis
- Visceral infiltration

Investigations

- **MRI (investigation of choice)**
- CT
- Biopsy.

Treatment

- Watching and waiting
- Surgical excision with safety margin (2.5 cm) $\pm$ postoperative radiotherapy to prevent recurrence.
- Anti-estrogens and NSAIDs.
- Chemotherapy to $\downarrow$ recurrence.

Bursae**Definition**

- It is a sac lined with synovial membrane containing fluid

Types

Anatomical	Adventitious
1- Around the knee: - pre-patellar (housemaid) - Infra-patellar (clergyman's knee) - Semi-membranous 2- Olecranon (students)	1- Over the head of shoulder (porters)
3- Subhyoid	2- Over external malleoli (trailors)
4- Radial bursa	3- Over the big toe (bunion)
5- Ulnar bursa	4- Over the amputation stump.

Bursitis

- 1- Traumatic: affect anatomical and adventitious
- 2- Infective:
 - Syphilis
 - Tuberculosis
 - Suppurative
- 3- Tumors: very rare

Semimembranous Bursitis**History (symptoms)**

- Adult age, presenting with painless or painful swelling in the popliteal fossa

On examination (signs)

- Cystic swelling on the upper medial part of the popliteal fossa, flaccid with flexion and tense with extension. (DD: Baker cyst $\rightarrow$ lies in the midline)

Differential diagnosis

- 1- Baker's cyst
- 2- Popliteal aneurysm
- 3- Other swellings in the popliteal fossa

Investigations

- Plain X-ray for the knee joint may be done to diagnosis associated lesion

Treatment

- Excision for primary type
- If secondary to knee effusion, treat the cause

Carotid Body Tumor

(Chemodectoma) (Potato tumor)

Definition

- It is rare slowly growing malignant tumor arising from the chemoreceptors presents at the bifurcation of the carotid artery.

- **Etiology:** slowly growing malignancy
- **C/P:** pulsating swelling at side of the neck
- **Invest.:** angiography
- **TTT:** excision

Clinical picture

- It presents at middle age as a slowly growing swelling which is smooth but may be lobular. The swelling moves from site to site but not vertical.
- It may be **pulsating** the pulsations are either transmitted from the carotid artery or expansile if the lesion is **highly vascular**.

Investigation

- **Angiography:** **widening the carotid artery bifurcation** which is characteristic sign.

Treatment

- Surgical excision with preservation of the internal carotid artery.
- If its preservation is not possible the artery should be replaced by a graft before excision to avoid interruption of blood flow to the brain.



Digital subtraction angiography

Clinical significance

- Carotid body tumor may present as a swelling in the pharynx, as tumor in the deep part of the parotid gland. That is why in case of any swelling in the oropharynx never to take a biopsy because CB tumor is highly vascular (patient become shocked).

Sternomastoid Tumor & Congenital Torticollis

Etiology

- Sternomastoid muscle develop by union of three **somites** each has its own blood supply.
- Sometimes at birth if interruption of the blood supply. This causes muscle infarction. The infarcted part becomes swollen, hence the name **sternomastoid tumor**. After a while the infarcted part will be replaced by fibrous tissue that will be contract causing congenital torticollis.

Clinical Picture

- At birth (or shortly afterwards) there is a painless firm swelling at the middle of the sternomastoid muscle.
- Later when torticollis develops the head will be tilted to the side of the lesion with the face looking to the opposite side.
- If the condition is neglected it leads to facial asymmetry.

DD

- **"Wry neck"** which is fibrositis causing spasm of the sternomastoid muscle. This condition is temporary lasting for a day or two and responds to anti-inflammatory drugs.

Treatment

- **Early after birth.** Attempt to prevent the development of the deformity by stretching and splinting of the neck may be done.
- **Established deformity.** Division of the sternomastoid at its lower part is done.

Surgical Skin Lesions

➡ Benign lesions of the skin & subcutaneous tissues:

1. Sebaceous cyst.
2. Dermoid cyst.
3. Callosity.
4. Corn.
5. Warts.
6. Papilloma.
7. Kerato-acanthoma.
8. Hemangioma.
9. Lymphangioma.
10. Lipoma.
11. Neurofibromatosis.
12. Navi.

➡ Precancerous skin lesions:

1. Squamous keratosis.
2. Bowen's disease.

➡ Malignant neoplasia of the skin:

1. Basal cell carcinoma.
2. Squamous cell carcinoma.
3. Melanoma.
4. Malignant skin tumors of vascular origins.

Benign lesions

1. Molluscum sebaceum (keratoacanthosis)

Definition

- It is keratinizing squamous cells which resolves spontaneously.

Incidence

- Middle age ♂ = ♀.

Etiology

- Prolonged exposure to sun.

Pathology

- Common in the face.

Clinical picture

- Firm, rounded, irreversible papule which enlarges rapidly over period of 8 wks. → producing rounded slightly umbilicated mass (< 2 cm in diameter), can be containing horny plug covered by a crust.
- Healing occurs by shedding of the horny core.
- Spontaneous healing takes about 6 m.
- Clinical importance:**
 - It mimics an epithelioma /or rodent ulcer.
 - Pathological examination: reveals abrupt transition from normal to hyperplastic epithelium.

2. Warts: "Verrucae vulgaris"

Cause Filterable virus

Site Common in the hands and soles of the feet. It can occur in any part of the body.

Clinical picture Small, horny with irregular surface, usually multiple

Treatment

- 1-Chemical paintings with glacial acetic acid
- 2-Electric cautery under local anesthesia
- 3-Surgical excision

3. Callosities

- It is an overgrowth of the epidermis with thickness of the horny layer.

Cause Prolonged friction or pressure

Clinical picture It appears as a horny yellowish plaque

Treatment Shaving + painting with salicylic acid and ether.

4. Corn

Cause

- Improper treatment of a callosity

Clinical picture

- It may be hard or soft. The central down-growth of a hard horny plug of epithelial cells into the dermis causing pain due to pressure on sensory nerve endings

Treatment

- Paint with 20% salicylic acid in collodion
- Excision (rare)

5. Papilloma (pedunculated papillomas "skin tags")

- It consists of central axis of connective tissue, blood vessels and lymphatics, surrounded by epithelium (squamous, transitional, cuboidal or columnar according to the site)

Common sites

- skin, kidney, colon, tongue and lips

Complications

- malignant changes
- Bleeding (hematuria, bleeding per rectum, nipple discharge)

Treatment

- Excision, cryosurgery or cautery

Precancerous skin lesions

1- Squamous keratosis (actinic / senile keratosis)

- It is a dry rough inelastic irregular pigmented area of the skin.
- Histology:
 - Area of hyperkeratosis, nuclear polymorphism, ↑ mitotic figures.
 - Squamous keratosis is the most important precursor of invasive cell carcinoma.

2- Bowen disease

- Arises in non-exposed skin.
- It is sharply, demarcated, rounded, reddish patches which enlarges slowly.
- There is marked epidermal hyperplasia usually misdiagnosed as psoriasis.

ULCERS

Definition

- Discontinuation of an epithelial surface.

Incidence

- It is common. Leg ulcers are the commonest chronic wounds in developed countries.

Etiology

A. Traumatic: could be self inflicted.

B. Inflammatory:

- Acute inflammation.
- Chronic inflammation
 - TB
 - Syphilis (1^{ry} chancre – 2^{ry} snail track ulcer – 3^{ry} gumma).

C. Neoplastic (Malignant ulcer):

- Squamous cell carcinoma.
- Basal cell carcinoma.
- sarcoma

D. Vascular:

- Arterial (ischemic)** → Large vessel (atherosclerosis)
→ Small vessels (DM)
- Venous** e.g. disease causing local venous hypertension e.g. varicosity.

Clinical picture

Symptoms

- Pain:** most non-specific ulcers are painful while specific ulcers are painless (except anal ulcers in homosexuals).
- Discharge:** e.g. purulent discharge if infected.

Signs

- Number.**
- Site:** e.g. venous ulcers occur specifically at the lower 1/3 of leg (gaiter area).
- Size:** malignant ulcers grow more rapidly than benign ulcers.
- Shape:** according to etiology, ulcers acquire specific shape.
- Edge.**
- Margin:** is the area between the edge and the normal skin.
- Floor:** can be seen.
- Base:** can be palpated.
- LN:** enlarges in infection and in squamous cell carcinoma.
- Vascular examination:** for arterial ischemia and venous varicosities.

Investigations

- A non-healing ulcer may require biopsy to exclude malignancy.

Treatment

➔ **Indications of surgical treatment:**

- Failure of medical treatment.
- Severe pain.

➔ **Lines of treatment:** skin grafts (meshed graft is better).

SINUSES and FISTULAS

Definition

- **Sinus** is a blind track leading from an epithelial surface into the surrounding tissues.
- **Fistula** is an abnormal communication between two epithelial-lined surfaces e.g. colocutaneous fistula.

Types

A. Congenital

- e.g. pre-auricular sinuses, branchial fistulas, tracheo-esophageal fistulas and arteriovenous fistulas.

B. Acquired

- Due to:
 - Inadequate drainage of an abscess e.g. perianal abscess leads to fistula.
 - Trauma or operation e.g. A-V fistula for renal dialysis.

Persistence of a sinus or fistula

- Due to:-
 - Presence of foreign body
 - Distal drainage
 - Distal obstruction
 - Presence of specific cause:
 - Specific infection (TB, actinomycosis)
 - Crohn's disease
 - Malignancy
 - Malnutrition
 - Usage of drugs e.g. corticosteroids

CHAPTER: 5

SALIVARY GLANDS

A- Non-neoplastic:

1. Congenital diseases.
2. Infections.
3. Salivary stones.
4. Salivary fistula.
5. Degenerative diseases.
6. Autoimmune salivary diseases.
7. Drug-induced endocrine & metabolic salivary gland enlargement.

B- Neoplastic

- 1- Benign
- 2- Malignant

Introduction

A. Surgical anatomy

- There are 3 pairs of main salivary glands (parotid, submandibular and sublingual glands) + small innumerable glands scattered in mucous membrane of mouth cavity (Nuhn & Blandin) (see for details in surgical anatomy book).
- **Contents of parotid gland:**
 - ⇒ Artery → External carotid A.
 - ⇒ Vein → Posterior facial V.
 - ⇒ Nerve → Facial M.
 - ⇒ L.N. → inside the capsule.
- **Parotid gland duct (Stenson's duct)** → opens opposite to the upper 2nd molar tooth in the vestibule of the mouth.

B. Surgical physiology

- 1- The characters of saliva secreted by parotid differ from those secreted from submandibular glands as parotid secretes serous fluid while submandibular secretes viscid fluid rich in calcium.
- 2- Functions of saliva are:
 - Food lubrication → swallowing
 - Cleaning of mouth.
 - Starch digestion by salivary amylase enzyme.
 - Mediates taste sensation.
- 3- Daily rate of salivary secretions about 1.5 liters/day.

Disease of Salivary Glands

Non-neoplastic

- 1- Congenital diseases.
- 2- Infections.
- 3- Salivary stones.
- 4- Salivary fistula.
- 5- Degenerative diseases.
- 6- Autoimmune salivary diseases.
- 7- Drug-induced endocrine and metabolic salivary gland enlargement.

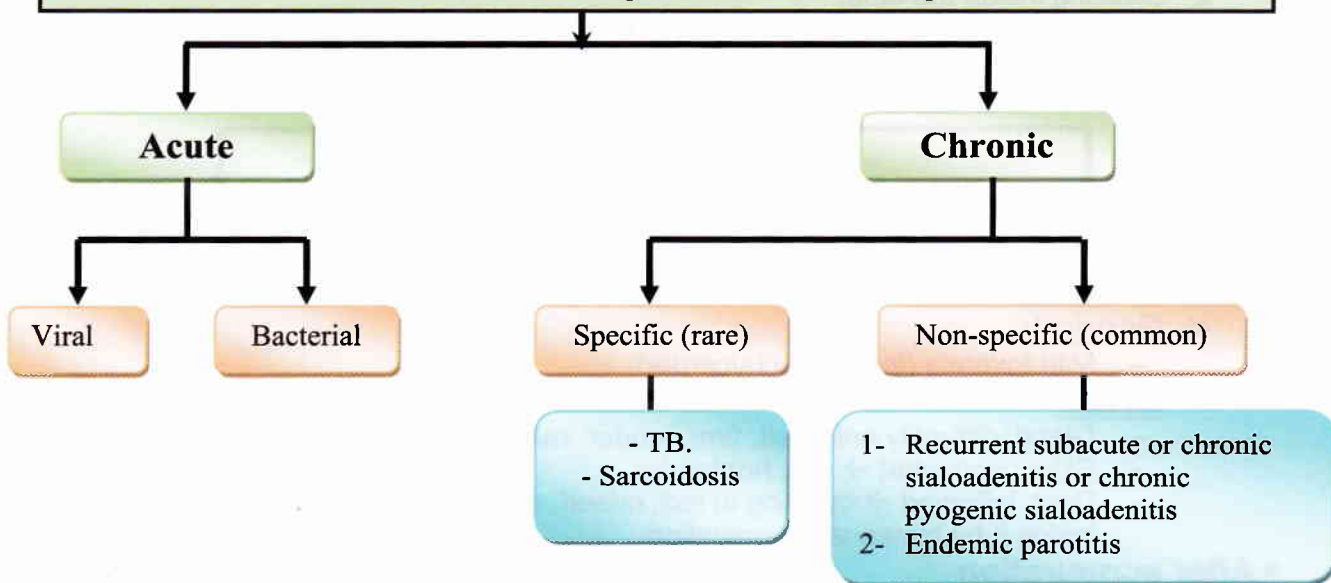
Neoplastic

- 1- Benign.
- 2- Malignant.

Congenital Diseases

- 1- Sialectasis (childhood type) (familial)
 - Degeneration of alveolar and duct system → dilatation → inadequate drainage → infection
 - Usually misdiagnoses in children as mumps but it differs from it being **unilateral and recurrent**.
 - It is self-limited after the age of 15 years and can be treated with antibiotics during the acute attack of infection.
- 2- Ectopic parotid tissue.
- 3- Cystic hygroma.

Infections (Sialoadenitis)



A-Acute Bacterial Sialoadenitis (acute suppurative sialoadenitis)

Definition

- Acute suppurative inflammation of salivary glands.

Etiology

Causative organism

- The commonest is staph. Aureus.
- Less commonly strept, **pneumococci**, etc....

Predisposing factors

1. Poor oral hygiene
2. It was common post-operative due to combination of dehydration and poor oral hygiene (due to **fasting + anesthetic medication** [atropine]) [parotid gland]

Precipitating factors

- Obstruction of salivary duct by **stone** or food particles

- **Etiology:** staph.aureus (P.F.= bad hygiene + post-op.)
- **C/P:** pain, swelling, lemon test, raising lobule of the ear
- **Comp.:** chronicity, abscess, stones, fistula, spread
- **Invest.:** as abscess + x-ray(stones)
- **TTT:** AAA + drainage(Hilton's)

Route of entry

- 1- Direct: along the duct from the mouth.
- 2- Blood born

Clinical picture

Symptoms

⇒ General:

- FAHM

⇒ Local

1- Pain:

In the side of the face, marked as the gland lies within tough fascia ↑ with eating

2- Swelling inside the face.

3- Disturbance of function: pain increases with ingestion of lemon or acidic juice (lemon test)



Signs

Before suppuration

After suppuration

▪ Before suppuration:

⇒ General:

- Mild toxemia (fever + tachycardia)

⇒ Local:

- Gland: diffusely enlarged, firm, tender, **raising lobule of the ear**.
- Skin: congested → red, hot tender.
- Duct: inflamed → opening in red, raised, edematous and stone may be felt inside it by bimanual examination

▪ After suppuration

⇒ General

- Marked toxemia → hectic fever + ↑ tachycardia

⇒ Local

- Gland: fluctuation is very late (tough fascia)
- Skin: Pitting edema + Marked tenderness
- Duct: pus exudates from the duct orifice after pressure on the gland ± stones felt on bimanual examination.

NB. Do not wait for fluctuation as fluctuation is very late (like Breast, Parotid, Pulb, Buttock)

Complications (infection)

- 1- Chronicity.
- 2- Abscess.
- 3- Stone.
- 4- Fistula
- 5- Spread of infection (ear, mastoid region and retropharyngeal space)

DD

- Acute lymphadenitis (multiple primary lesion, it can be rolled over the mandible)

Investigations (infection)

- 1- CBC → leucocytosis.
- 2- ↑ESR, ↑CRP.
- 3- Culture and sensitivity of purulent discharge.
- 4- X-ray → for stone.

Treatment (infection)

A- Prophylactic

B- Curative

A. Prophylactic

- Avoid predisposing factors.

B. Curative

- 1- **Initially** → 3 As (antibiotics, analgesics, antipyretics) + hot fomentation. The best antibiotic is **Clindamycin** as it attains the highest concentration in saliva.
- 2- **Indications of surgery:**
 - Failure to respond to conservative treatment within 48 hours.
 - Evidence of abscess formation.

⇒ Surgical drainage:

By Hilton's technique if parotid abscess.

- 1- The skin is incised vertically (blaire's incision) under general anesthesia from lobule of ear to angle of mandible then incise deep fascia transversally.
- 2- Then blunt tipped forceps is introduced closed at the suspected area then opened gently along the direction of facial nerve branches to avoid their injury.
- 3- Sample of pus is taken for C&S.
- 4- Rubber drain is inserted and kept for few days till discharge stops.

⇒ Surgical drainage of submandibular abscess

- 1- Skin incision parallel to the lower border of mandible 1.5 cm below and in front of angle of mandible to avoid injury of mandibular branch of facial nerve.
- 2- Incise deep fascia and push sinus forceps parallel to branches of facial nerve (Hilton technique)

⇒ Complications of drainage (3F):

- 1- Salivary fistula.
- 2- Facial nerve injury.
- 3- Frey's postoperative syndrome → excessive sweating + flushing when eating.

Acute Viral Parotitis (acute non-suppurative parotitis) (mumps)

- Caused by mumps, presents in children (15 years), with incubation period (15 days), bilateral painful parotid gland swelling with fever and it is self-limited.
- **Complications:**
 - Pancreatitis, encephalitis, orchitis and oophoritis

Chronic Sialoadenitis

Pyogenic (recurrent subacute)

Endemic parotitis

B-Recurrent Subacute and Chronic Pyogenic Sialoadenitis

Incidence

- Submandibular > parotid gland.
- **Predisposing factors:**
 - 1- Following acute due to persistence of the same predisposing factors or inadequate treatment.
 - 2- Partial duct obstruction by stone or stricture.
 - 3- Sialectasis.
 - 4- Autoimmune diseases of salivary gland.

Clinical picture

- Chronic sialoadenitis of submandibular salivary gland presents with swelling in digastric triangle characterized by:
 1. History of pain and increase size of swelling during eating.
 2. Swelling is solitary, can not be rolled over the edge of mandible.
 3. Inspection of mouth floor may reveal redness of duct orifice
 4. Bimanual palpation reveals that swelling is filling the floor of the mouth.

The previous characters of submandibular salivary gland swelling differentiate it from submandibular LNs swelling.

Investigations

- 1- Plain X-ray → stone
- 2- Sialogram → sialectasis → dilated duct.

Treatment

- 1- Treatment of predisposing factors + antibiotics + mouth wash.
- 2- Surgery for chronic submandibular sialoadenitis → submandibular sialodentomy.

2-Chronic endemic parotitis (RES hyperplasia)

- Due to chronic parasitic infestation = hypoproteinemia e.g. liver cirrhosis and malnutrition.
- Presents with bilateral soft painless enlargement of the parotid gland.

B- Chronic specific sialoadenitis (TB, Sarcoidosis)

- Rare, if they occur, they usually affect parotid lymph nodes rather than the gland stroma.

Salivary Stone (Sialolithiasis)(Salivary Colic)

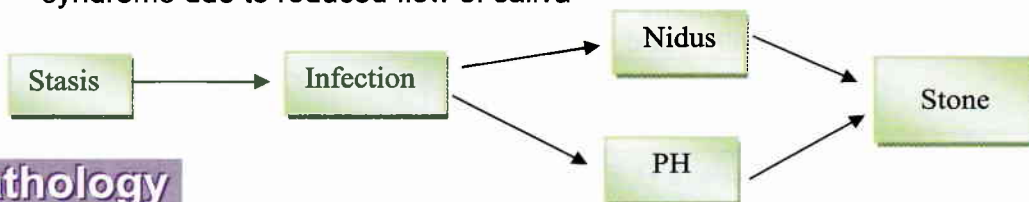
Incidence

- **Submandibular > parotid (50:1) due to:**
 - 1- Gland secretions are more viscid with high calcium concentration.
 - 2- Duct ascends upwards → inadequate drainage.
 - 3- Orifice lies in the floor of the mouth → liable to be blocked by food particles.

- **Etiology:** infection, stasis
- **C/P:** asymptomatic, pain
- **Comp.:** obst.(sialadenitis), fistula
- **Invest.:** x-ray, sialography, U/S
- **TTT:** according to site of the stone

Predisposing factors

- 1- **Infection:** (chronic sialadenitis) due to change of pH of saliva + provide nidus for stone (pus cells)
- 2- **Stasis:** causes stagnation and infection. So stone complicated Sjogren's syndrome due to reduced flow of saliva



Pathology

- 1- **Number:** single or multiple.
- 2- **Site:** inside gland or duct.
- 3- **Nature:** Calcium + magnesium + phosphorus + carbonate

Complications

- 1- Obstruction of duct → sialectasis + sialadenitis.
- 2- Salivary fistula.

Clinical picture

Symptoms

- **Mainly asymptomatic** or may present with:
 1. As chronic sialadenitis (*Episodes of pain followed by relief*) especially after eating.
 2. Usually in the side of the face.

Signs

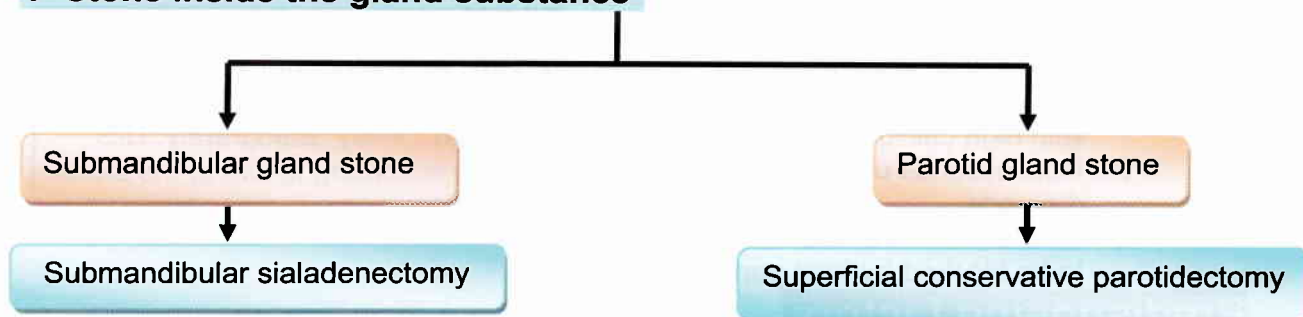
- Especially if acute on top of chronic sialoadentitis (**see before**).

Investigations

- 1- Plain X-ray (occlusal view) → stone in submandibular gland is radio-opaque in 80% of cases due to high calcium concentration
- 2- Sialography → best in parotid stones as they are radiolucent. They appear as filling-defect or a block to sialography + dilated ducts
- 3- Ultrasound → shows the stone as echogenic structure inside the gland with acoustic shadowing.

Treatment

Surgical (according to the site of stone)

1- Stone inside the gland substance**2- Stone inside the duct**

- Peeping stone from orifice → meatotomy.
- Stone in duct → removed through mouth under local anesthesia.
- Submandibular sialoadenectomy → if recurrent or associated with gland stroma.

Usually difficult (why?) as recurrent inflammation makes identification of facial nerve very difficult

Salivary Fistula**Etiology**

Stone → Abscess → Fistula

1- Trauma:

- Due to accidental trauma to parotid gland or duct or less commonly surgical trauma.

2- Inflammatory:

- After drainage or spontaneous rupture of salivary gland abscess.

3- Neoplastic:

- Due to malignant tumor infiltrating skin.

- **Etiology:** trauma, infection, neoplastic
- **Types:** external, internal
- **C/P:** discharge, eczema
- **Invest.:** sialogram
- **TTT:** according to site

Types**1- External:** opens on the skin (requires treatment)**2- Internal**

- Opens on mucous membrane of mouth due to ulceration of duct stone. through mouth cavity (it is harmless condition that **requires no treatment**)

Why fistula that arises from the duct is unlikely to heal?

Because of high rate of salivary flow

Clinical Picture

- History of the cause.
- Watery discharge from opening of fistula if external type especially during smell or taste of food.
- Eczema of skin around fistula opening.

DD

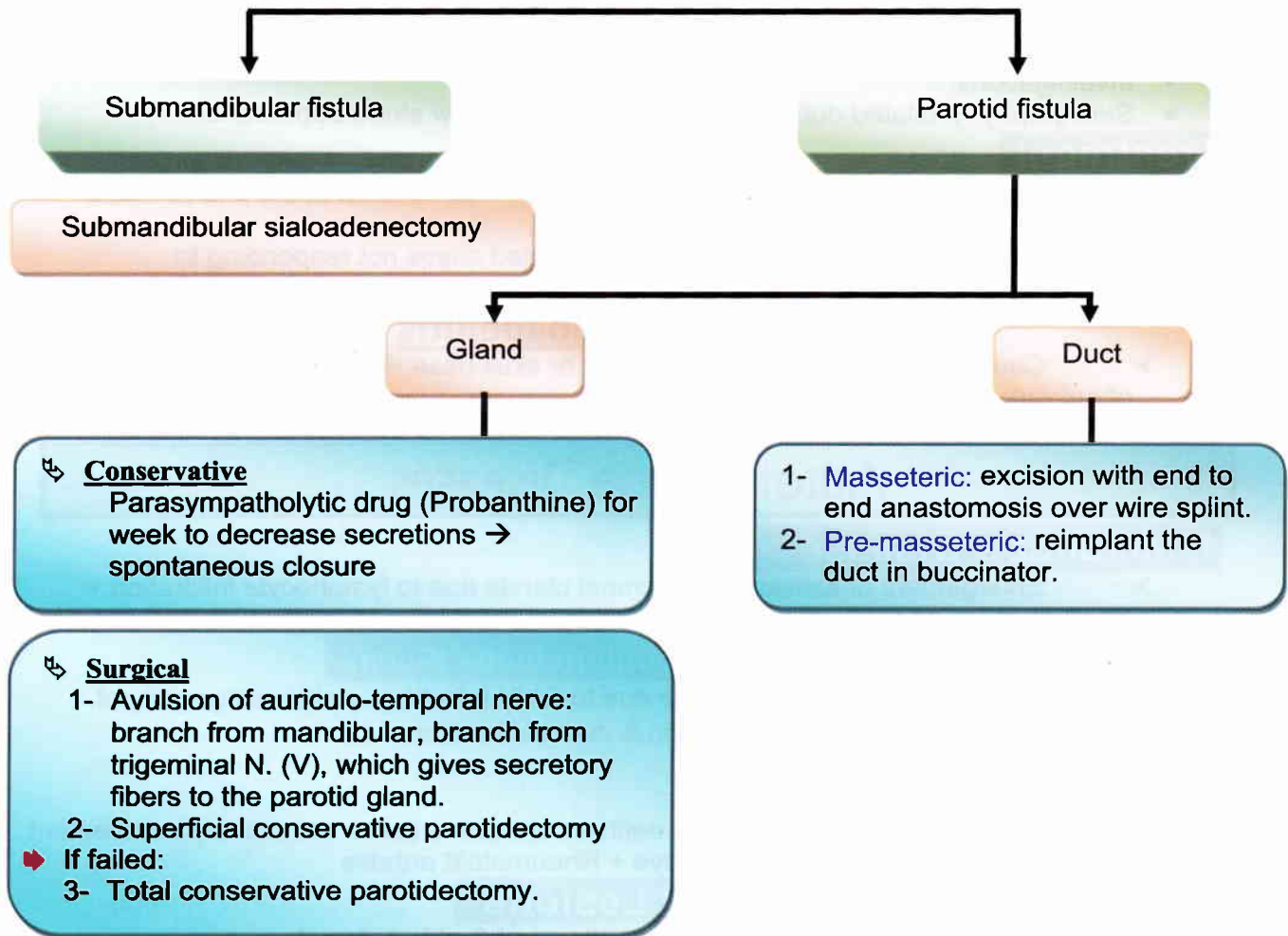
- Fistula in the face (salivary fistula, dental, osteomyelitis of the mandible and pre-auricular sinus)

Investigations

- Sialogram to exclude distal duct obstruction by stone or stricture (0.5 – 2ml of radioopaque as lipiodol or hypaque "sodium diatrizole").

Treatment

- The distal part of the duct is cannulated from the mouth & a trial is made to repair it without tension.
- If failed, surgical treatment according to site of fistula.



Degenerative Diseases

1. Sialectasis (adult type)

Definition

- Abnormal dilatation of small branches of salivary ducts and alveoli secondary to Sjogren's syndrome or occupational increase of intra-oral pressure as glass blowing
- Degeneration of acini → cystic dilatation → stasis → repeated sialoadenitis.

Presentation

- Recurrent attacks of sialoadenitis.
- Investigations.
- Sialography → dilated ducts and branches giving snow storm appearance.

Treatment

- 1- **Conservative:** massage + antibiotic for infection + citrus drink at the end of meals to stimulate salivary flow.
- 2- **Surgery:** surgical excision of gland in complicated cases not responding to conservative treatment.

2. Radiation Sialoadenitis

- Caused by radiation to nasopharynx or skull base → temporary suppression of salivary secretion.
- Treated by administration of sialogogue as citrus fluid.

Autoimmune Diseases

1. Mikulicz's Disease

- Enlargement of salivary and lacrimal glands due to lymphocyte infiltration + dryness of mouth.

2. Sjogren's Disease (Keratoconjunctivitis Sicca)

- **Common in females**, may be due to CMV infection → alter antigenicity of salivary duct → lymphocyte infiltration & duct obliteration.
- ↑ risk of lymphoma 44 times.
- **Types:**
 - a- Primary: dry mouth + dry eye neither connective tissue nor salivary involvement
 - b- Secondary: dry mouth + dry eye + Rheumatoid arthritis

3. Benign Lymphoepithelial Lesions

- Uncommon disease, affecting middle aged & elderly females.
- Characterized by diffuse enlargement of salivary glands especially submandibular and parotid due to progressive lymphocytic infiltration.
- Prelymphomatous potential 20%.

Diagnosis of autoimmune diseases

- Lip biopsy that includes the minor salivary glands.
- Parotid sialography shows the nonspecific appearance of narrowed ducts and sometimes punctate sialectasis.

D.D. of bilateral enlargement of parotid gland

1. Endemic parotitis.
2. Autoimmune (Sjogren \$).
3. Monomorphic adenoma.

Treatment

- Instillation of artificial tears that are made up of methylcellulose helps to combat eye dryness.
- Meticulous oral hygiene.
- If a patient with these autoimmune salivary diseases develops a palpable mass in the parotid, needle biopsy should be performed. If this proves to be a lymphoma, the gland is removed, the disease staged, and treatment is continued with radio and/or chemotherapy.

Drug-induced, metabolic and endocrine salivary gland enlargement

- Drug-induced enlargement of the salivary glands has been reported with sulfoxazole, phenylbutazone, iodide containing compounds, thiouracils, hypotensive drugs, and contraceptive pills.
- Metabolic and endocrine causes include liver cirrhosis, diabetes, alcoholism, malnutrition, and ovarian, pancreatic, or thyroid insufficiency.

Syndromes Related to Salivary Glands

The auriculotemporal syndrome (Frey's syndrome) (gustatory syndrome)

- ☒ This condition follows surgery in the region of the parotid gland or tempromandibular joint.
- ☒ Also, it may follow accidental injury of the previous sites

Pathogenesis

- (cross regeneration of parasympathetic and sympathetic fibers)
- After injury of auriculo-temporal nerve, post ganglionic parasympathetic fibers from the otic ganglion become united to the sympathetic nerves from superior cervical ganglion destined to supply the vessels and sweat glands of the skin

Clinical feature

- Flushing and sweating of the skin innervated by auriculotemporal nerve when even the salivation is stimulated

Salivary Neoplasms (Parotid Tumors)

- Salivary neoplasms constitute 5% of head and neck tumors & 1 – 2 of body neoplasms.
 - The majority of these neoplasms are benign; most commonly arise in the parotid gland.
 - The majority of this neoplasm arise from the parotid, the incidence of malignancy varies inversely with the size of the gland:-
 - 25% of the parotid gland.
 - 40% of submandibular gland.
 - 70% of sublingual gland.
 - 90% of minor salivary gland.
- } Swellings are malignant
- The larger the gland, the more common the tumor incidence, the smaller the gland the, the higher the malignancy rate.

Benign	Malignant
1- Pleomorphic adenoma	1- Mucoepidermoid carcinoma
2- Monomorphic adenoma	2- Adenoid cystic carcinoma (cylindroma)
3- Wharthin's tumor	3- Acinic cell carcinoma
4- Adenolymphoma	4- Adenocarcinoma
5- Oncocytoma.	5- Carcinoma ex pleomorphic adenoma
	6- Lymphoma

Pleomorphic Adenoma (Mixed Tumor)

Incidence

- Commonest salivary tumor, more common in males.
- Commonest in the parotid, less in submandibular gland.

Pathology

- Benign tumor.

Site

- Usually from superficial part of parotid gland.

Macroscopic

- Firm, bossy, irregular, lobulated, encapsulated with grayish white cut surface and strands of tumor cells tend to penetrate the capsule (**incomplete capsule**) and may extend beyond the main limits of the mass (**multicentric**) so enucleation of the tumor carries high risk of **recurrence**.

Microscopic

- It was referred to in the past as mixed salivary tumor because of its histological appearance.
- **Cells:** epithelial cells arranged in sheets and duct-like structures.
- **Matrix:** blue-stained mucinous material (pseudo-cartilagenous).

Clinical features

Symptoms

- Painless, slowly growing swelling in the side of the face.

Signs

- Site: in parotid region.
- Size: variable.
- Shape: irregular.
- Surface: lobulated.
- Consistency: variable (**firm or cystic but never hard**).
- Mobility: freely mobile (not attached to skin, muscles or bones).
- Special character: elevating lobule of the ear.
- Spread: no cervical LNs enlargement or facial nerve infiltration.

DD

- See later.

Complications

- 1- Disfigurement.
- 2- Malignant transformation after > 10 years is rare (2-3%).

Investigations

- 1- CT scan for assessment of tumors arising from deep part of the parotid.
- 2- Tc99 scan: cold spot (avascular)
- 3- Open biopsy is not advised to avoid fistula, facial N. injury & spillage of the tumor.
- 4- Fine needle biopsy may be required.

- **Etiology:** benign tumor
- **C/P:** painless mass raising ear lobule
- **Comp.:** cosmetic, malignancy
- **Invest.:** CT scan, FNAB
- **TTT:** according to the gland



Treatment

- The tumor should be excised with safety margin (less recurrence rate than enucleation)

1- Benign tumor in parotid:

- Conservative superficial parotidectomy
 - All parotid tissue that is superficial to the nerve and its branches is excised
- Total conservative parotidectomy:
 - Removal of both superficial and deep lobes with preservation of facial nerve (indicated for tumors of the deep lobe and for recurrent tumors)

2- Benign tumor in submandibular gland:

- Treatment is submandibular sialadenectomy and take care not to injure 3 nerves:
 - 1- Cervical & mandibular division of facial nerve → protected by making an incision parallel to and 2 cm from lower border of mandible.
 - 2- Lingual and hypoglossal nerves are at risk during excision of deep part of the gland → protected by exposure of these nerves.
- **Minor salivary gland tumors (rare) Simple excision with safety margin**

What to do if facial nerve is injured?

In case of accidental nerve injury → immediate repair by microsurgical techniques either by:

- 1- Direct suturing
- 2- Nerve graft taken from great auricular nerve.
- 3- Hypoglossal sling: anastomosing peripheral branches of facial nerve to hypoglossal nerve

Monomorphic Adenoma (Adenolymphoma) (Warthin's tumor) (Papillary Cystadenoma Lymphatosum)

Incidence

- 10% of parotid tumors and bilateral in 10% of cases especially in old age and smokers.

- **Etiology:** benign tumor
- **C/P:** painless mass not elevating ear lobule
- **Invest.:** CT scan
- **TTT:** Cons. sup. parotidectomy

Pathology (papillary Cystadenoma Lymphatosum)

Site

- Superficial part and lower lobe of parotid gland (it is thought to arise from heteropic salivary tissue in parotid LNs)

Macroscopic

- Cystic, encapsulated benign tumor

Microscopic

- Columnar epithelium surrounded by lymphoid tissue

Clinical picture

Symptoms

- Elderly male > 50 years, presents with painless, slowly growing swelling in side of the face.

Signs

- Same as pleomorphic adenoma but always cystic or soft in consistency, in front of tragus of ear, not elevating lobule of the ear, bilateral, never to turn malignant.

DD (swelling in the parotid region)

- **Parietal lesions:** e.g. lipoma, neurofibroma,etc.
- **Musculoskeletal:**
 - ✓ **Muscular (from masseter muscle):** fibrosarcoma, masseter muscle hypertrophy (usually bilateral)
 - ✓ **Bony (from the ramus of mandible),** Burkitt's lymphoma
- **Pre-auricular LN swellings:**
 - ✓ **Lymphadenitis:** acute & chronic (non-specific & specific e.g. TB lymphadenitis).
 - ✓ **Malignancy:** lymphoma & metastatic carcinoma.
- **Parotid gland:**
 - ✓ **Inflammation:**
 - Acute (viral, bacterial).
 - Chronic (TB, Sarcoidosis).
 - ✓ **Autoimmune:**
 - Sjogren's syndrome.
 - Benign lymphoepithelial lesion.
 - ✓ **Tumors:**
 - **Benign:**
 - Pleomorphic adenoma
 - Adenolymphoma
 - Oncocytoma.
 - Monomorphic adenoma.
 - **Malignant:**
 - Mucoepidermoid carcinoma
 - Adenoid cystic carcinoma
 - acinic cell carcinoma
 - Carcinoma ex pleomorphic adenoma.

Investigations

- 1- Tc99 scan: hot spot
- 2- CT scan

Treatment

- Conservative superficial parotidectomy

Why it is mandatory to examine oropharynx in case of parotid tumor?

- Because tumors arising in the deep part of the parotid, may bulge in the oropharynx behind the tonsils so it is important to examine mouth cavity.
- Or it may be a carotid body tumor. So, if there is any swelling in the oropharynx → don't take a biopsy, it may be a carotid body tumor which is highly vascular.

Malignant Neoplasms of Salivary Glands (Carcinoma of salivary glands)

Etiology

- 1- De-novo.
- 2- On top of mixed parotid tumor.

Pathology

Macroscopic

- Non-capsulated infiltrating tumor with grayish areas of hemorrhage and necrosis

Microscopic

a. Mucopidermoid carcinoma:

- The commonest type, arises from the duct epithelium, consists of sheets of columnar (mucoïd) and squamous (epidermoid) cells of 3 grades of differentiation (low, intermediate, high) the low grade is the commonest & can affect children.

b. Adenoid cystic carcinoma (Cylindroma)

- The commonest malignancy affecting minor salivary glands characterized by slow rate of growth & peri-neural spread (painful) making tumor resection usually incomplete & tends to recur.
- (Swiss-cheese pattern) → composed of myoepithelial cells (basophilic) + epithelial cells (eosinophilic) → cribriform (Swiss-cheeses appearance)

c. Acinic cell carcinoma

- Composed of serous acini (so occur only in parotid gland)

d. Adenocarcinoma (bad prognosis):

- Rare, 3 basic histological patterns (tubular, papillary and undifferentiated)

e. Carcinoma ex pleomorphic adenoma

f. Undifferentiated (the more the undifferentiation the worse the prognosis)

Spread

1- Direct:

- To mandible, masseter, facial nerve in parotid tumors, lingual and hypoglossal nerve in submandibular tumors

2- Lymphatic:

- To parotid and submandibular LNs → upper deep cervical LNs

3- Blood spread

- Rare and late to lung and bone.

Complications

- 1- Ulceration.
- 2- Infection.
- 3- Hemorrhage.
- 4- Nerve palsy.

- **C/P:** pain, swelling, LN, comp.
- **Comp.:** ulceration, inf., hge., n.palsy
- **Invest.:** as any cancer
- **TTT:** -Operable: radical ttt:
 - 1-total parotidectomy
 - 2- commando op.
- Inoperable: palliative ttt

Clinical Picture

Symptoms

- 1- **Pain:** may radiate to the ear, increase by mastication (through Arnold branch of vagus N.)
- 2- **Swelling:** slowly growing on the side of the face.
- 3- **Disturbance of function = complications (e.g. facial N. paralysis).**

Signs

- 1- **Swelling:**
 - Firm or hard in consistency.
 - Irregular in shape.
 - Nodular surface.
 - Ill-defined edge.
 - Infiltration of skin, vessels, nerve, L.N.
- 2- **Cervical LNs:**
 - Enlarged, stony hard, mobile then fixed.

Investigations

For diagnosis

- 1- Tc99 scan → cold spot
- 2- Biopsy → from the gland

For staging

- 1- CT scan to determine local extent of tumor
- 2- FNAB from LNs
- 3- Metastatic work up

Open surgical biopsies are contraindicated in major salivary gland neoplasm, FNABC is safer

Treatment

Operable

⇒ **According to site of tumor:**

- 1- **Cancer in parotid gland:**
 - total radical parotidectomy + total block dissection of the neck LNs + postoperative radiotherapy to decrease recurrence
- 2- **Carcinoma in submandibular gland:**
 - Commando operation (total radical submandibular sialadenectomy + hemimandibulectomy with adjacent part of tongue in continuity + block dissection of the neck LNs)

- Facial nerve should be sacrificed and if injured repair by microsurgical technique (see before)
- In low grade mucoepidermoid carcinoma an attempt to preserve facial N. is warranted.

Inoperable

- Palliative resection + radiotherapy.

Tumors that Clinically Malignant

- If pathological diagnosis not obtained → intraoperative frozen section:
 - High grade & -ve L.N. → block neck dissection.
 - Low grade & -ve L.N. → no block neck dissection.
- Postoperative radiotherapy of limited value, but it is administrated as postoperative adjuvant in high grade malignancies.

TNM staging

TX	primary tumor cannot be evaluated
T0	No evidence of a tumor is found
T1	Non-invasive tumor , <2 cm.
T2	Non-invasive tumor, 2 - 4 cm
T3	4 -6 cm , spread beyond the salivary glands, but does not affect the facial nerve
T4a	invades the skin, jaw, ear canal, and/or facial nerve
T4b	The tumor invades the skull and/or the nearby bones and/or arteries

NX	Regional lymph nodes cannot be evaluated
N0	There is no evidence of cancer in the regional nodes
N1	Spread to a single node on the same side
N2	More than one lymph node on the same side , or on either side of the body , < 6 cm
N3	Cancer found in lymph nodes is larger than 6 cm
MX	Distant metastasis cannot be evaluated
M0	No distant metastasis
M1	Distant metastasis to other parts of the body

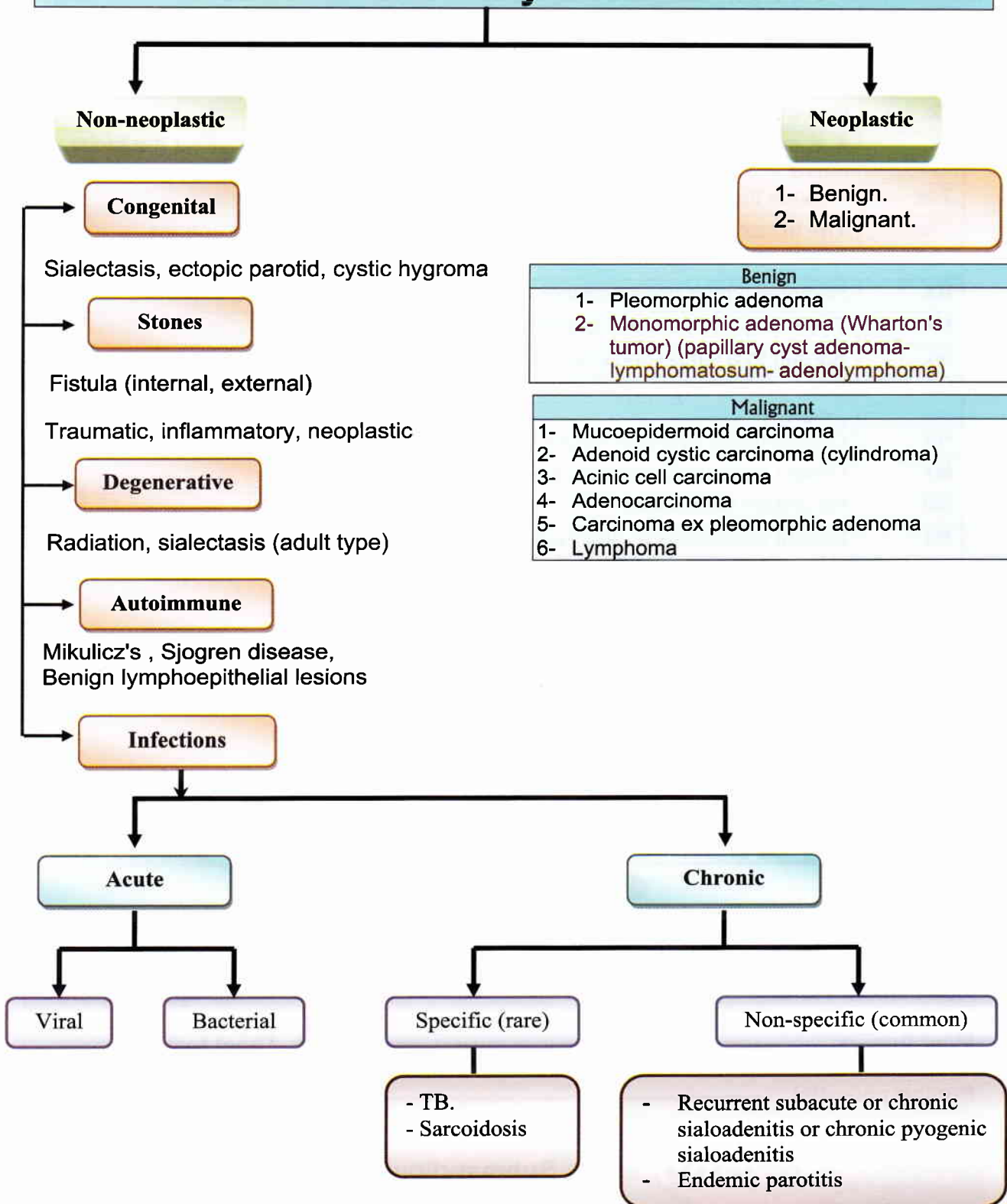
- ❖ **Stage I:** T1, T2, N0, M0.
- ❖ **Stage II:** T3, N0, M0.
- ❖ **Stage III:** T1, T2 ,N1, M0.
- ❖ **Stage IV:**
 - **Stage IVa:**
 - T3,4 N0,1,2, M0.
 - **Stage IVB:**
 - T, N2, N3, M0.
 - **Stage IVC:**
 - M1.

Summary of incidence of affection of different glands in different diseases

Serous secretions	→	Mucinous secretions
Radiolucent stone	→	Radio-opaque stone
Most tumors	→	Least tumors
Benign potential	→	Malignant potential

Parotid**Submandibular****Sublingual**

Scheme of Salivary Gland Diseases



CHAPTER: 6

SURGICAL INFECTIONS

- 1- Acute abscess
- 2- Cellulitis
- 3- Erysipelas
- 4- Boil
- 5- Carbuncle
- 6- Ludwig's angina
- 7- Impetigo
- 8- Hand infections
 - General scheme
 - Acute paronychia
 - Chronic paronychia
 - Pulp space infection
 - Suppurative tenosynovitis
 - Ulnar bursitis
 - Radial bursitis
 - Web space infection
 - Thenar space infection
 - Superficial Mid palmar space infection
 - Deep mid palmar space infection
 - Subcuticular whitlow
 - Ingrowing toe nail
- 9- Tetanus
- 10- Gas gangrene
- 11- Actinomycosis
- 12- Necrotizing fasciitis
- 13- Nocardiosis
- 14- Surgical site infection
- 15- Antibiotics in surgery

Surgical Infections

Pathogenesis

☞ **3 elements are important for surgical infections to take place:**

- A. Infective agents.
- B. Susceptible host.
- C. A closed local space which is suitable for infection to occur.

A. Infective agents

- The development of infection is favored by the number and the virulence of the invading organisms.
- Involved organisms may be:
 - a. **Aerobic**: as streptococci, staphylococci, klebsiella and E. coli.
 - b. **Anaerobic**: as Bacteroides and Clostridium.
- Virulent organisms are highly invasive and cause primary infections.
- **Infective organisms may be:**
 - **Endogenous** from within the body, as in gut perforation producing peritonitis.
 - **Exogenous** (hospital acquired → called secondary or nosocomial infection).

B. Susceptible host

- The host response to bacterial invasion can be reduced by several conditions as:
 1. Malnutrition.
 2. Metabolic disease (D.M. and Uremia).
 3. Immune suppression e.g. steroids, chemotherapy, radiotherapy and AIDS.

C. Closed suitable space

- Most surgical infections start in a susceptible poorly vascularized tissue such as a wound or a natural space with narrow outlet which is prone to obstruction as gall bladder and vermiform appendix.
- **The risk of infection in wound is increased by:**
 1. Poor surgical manipulation with devitalization of the tissues → leaving a large dead space with hematoma formation.
 2. Bad general condition (e.g. D.M., senility).
 3. Bad hygiene.
 4. Foreign body.
 5. Ischemia.

Scheme for infection:

- **Definition.**
- **Etiology:** causative org., PDF, route.
- **Pathology.**
- **C/P:** symptoms: general & local
Signs: general & local.
- **Complications.**
- **Investigations.**
- **Treatment:** medical & surgical.

Acute Abscess

Definition

- A localized suppurative inflammation.

Etiology

Organism

- Usually staph (coagulase +ve), especially in skin & neck abscess.
- Less commonly strept, E. coli.
- The commonest organism in abdominal abscess is E.coli

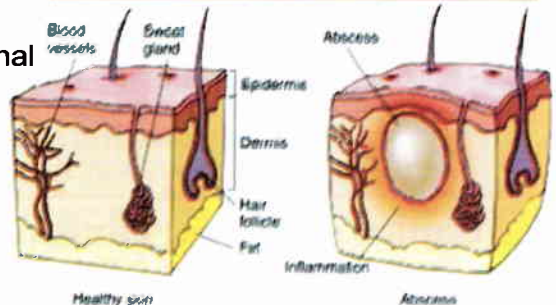
- **Etiology:** -Skin: staph. Aureus
-Abdomen: E.coli
- **C/P:** FAHM, pain, swelling, LN
- **Comp.:** general, local
- **Invest.:** CBC, C&S, acc. to site
- **TTT:** AAA + incision & drainage

Predisposing factors

- Poor general resistance, DM, senility.
- Bad hygiene.

Route of infection

1. **Direct** → through a wound or ulcer.
2. **Blood** → from a septic focus (bacteremia).
3. **Lymphatic** → from an infected area.
4. **Natural passages** e.g. airways: lung abscess.



Pathology

The abscess formed of 3 zones.

1. Central zone

- Liquefaction, by dead WBCs.
- Pus is formed of: necrotic tissue, inflammatory exudates, organism and dead leucocytes.

2. Intermediate zone

- Formed of granulation tissue.
- Appear to secrete pus, hence the old term pyogenic membrane.

3. Peripheral zone

- Of hyperemia.

Clinical Picture

Symptoms

⇒ **General:** (FAHM)...etc.

⇒ **Local**

- Pain:

○ At first dull aching pain before pus collection then → throbbing with collection of pus.

○ Decreased by elevation of the part.

- Swelling: → due to edema of the tissues.

- Loss of function → due to pain.

Signs

⇒ **General:** fever (hectic or spiky), tachycardia.

⇒ **Local:**

- Swelling → Hot & red, Tender.
→ At first indurated then soften at center.
→ After 2-5 days pus points & ruptures.
- LNs → **E**nlarged, **E**lastic, **T**ender, **M**obile.
- Fluctuation is very late & should not be waited in the breast, prostate, parotid, perineum, pulp space (5Ps) & Ludwig's angina.

- An abscess starts as painful tender mass covered by skin, enlarged L.N. & FAHM.
- Once pus formed:
 1. Pain become throbbing.
 2. Fever become hectic.
 3. Covering skin shows pitting edema.
 4. Fluctuation is a late sign (only in superficial abscess, but false sense of fluctuation in breast abscess & difficult to be elicited in parotid & gluteal abscess).
 5. Shooting leucocytosis with shift to the left.

Examples of special clinical picture according to site

- Brain abscess: clinical picture of increased ICT.
- Lung abscess: pus on postural drainage.

Complications & Fate**General**

- **Toxemia** → absorption of bacterial toxins into the circulation.
- **Bacteremia** → transient presence of bacteria into the blood. It may be dangerous in those with cardiac or orthopedic prosthesis → prophylactic antibiotics before minor procedures.
- **Septicemia** → persistent bacteremia **multiplying** in the blood, toxins.
- **Pyemia** → septic emboli released from an infected thrombus.

Local

1. **Pointing & rupture (Commonest sequel)**
 - The pus passes along the line of the least resistance.
 - It points on the skin, mucous membrane or serous surface.
 - It ruptures & discharges its contents.
2. **Chronicity** due to:
 - Organism tends to be chronic.
 - Persistence of predisposing factors
 - Inadequate drainage & treatment.
3. **Spread:**
 - Cellulitis.
 - Lymphangitis.
 - Lymphadenitis.
4. **Sinus** (epithelialized tract connected to skin surface) & **Fistula** (pathological tract between 2 epithelial surfaces) formation, especially with actinomycosis and mycobacterium.
5. **Antibioma (it is a chronic abscess not a tumor) common in breast.**
 - If sterile pus is formed, not drained.

Investigations

- **CBC:** leucocytosis.
- **Culture and sensitivity.**
- **Plain X-ray** e.g. lung abscess.
- **U/S** e.g. amoebic liver abscess.
- **CT and MRI** e.g. brain abscess.
- **Aspiration can be done guided by X-ray, U/S, CT or MRI.**

Treatment

General

- Analgesics, antipyretic and antibiotic (even parenteral in severe cases) (**AAA**).
- Antibiotics are not alternative to drainage, but given when infection is not under control and spreading.

Local

1. **Rest of the affected organ** +TTT of predisposing factors.
2. **Hot fomentation** (hyperemia brings more antibiotics & antibodies).
3. **Incision & drainage:**
 - Under general anesthesia or local regional anesthesia.
 - The incision should be:
 - Long & Dependent → allow good drainage.
 - Never cross a skin crease.
 - Parallel to important structures e.g. nerves & vessels.
 - After incision → introduce a finger (or mosquito) to break all septa.
 - Packing for 48 hours for hemostasis.
 - Later on → dressing without packing every day.
 - After pus discharge becomes minimal → dressing every few days until complete healing.

Hilton's methods

- In areas with important structures (e.g. axilla, neck, parotid).
- The skin is only incised.
- Abscess cavity is entered by closed blades of artery forceps.
- Then the blades are opened widely.

Aspiration used in

- Amebic liver abscess - Brain abscess - Cold abscess.

Chronic abscess:

- Thin walled → incision & drainage - Thick walled → excision

Key points

1. Antiboma: not a tumor and common in the breast.
2. Abscess which we don't wait for fluctuation (5Ps).
3. How to drain abscess and Hilton technique.

Cellulitis

Definition

- Is an invasive non-suppurative infection of loose C.T. (although PMNL are abundant, there is no pus formation).

Etiology

- **Etiology:** strept.
- **C/P:** as before + ill-defined edge
- **Comp.:** general, local
- **Invest.:** CBC, C&S
- **TTT:** AAA + ttt of comp.

Organism

- Usually strept → produces hyaluronidase & fibrinolysin → spread.

Predisposing Factors & Route

- See before.
- Scratch or prick.

Clinical Picture

Symptoms

See before

Signs

- ⇒ **General:** see before.
- ⇒ **Local**
 - Tender, dusky red, hot area with indurated edge (never affect ear pinna)
 - LNs → enlarged, elastic, tender, mobile.
 - Red strikes of lymphangitis
 - Spreads rapidly with ill defined edge.
 - No suppuration.



Complications

- General + chronicity + pus loculus + post-streptococcal glomerulonephritis + scarlet fever + SIRS (common).

Post streptococcal GN usually follows skin infection while post-streptococcal rheumatic fever usually follows tonsillitis.

D.D.

- Contact allergy.
- DVT
- Chemical infection (at the site of drug injection).

Investigations

- CBC: leukocytosis.
- Blood cultures are often negative.

Treatment

- See before (AAA + rest + hot fomentations).
- Antibiotic of choice is penicillin (benzyl penicillin 600-1200 mg i.v./6hours). Erythromycin is used as alternative in patients allergic to penicillin.
- If no response after 48 hrs → either diagnosis of cellulitis is doubted, an abscess is suspected or resistant organisms are involved.

Key points

1. Non-suppurative and poorly localized.
2. SIRS is common + GN and scarlet fever.
3. Antibiotic of choice is penicillin.

Erysipelas

Definition

- Non suppurative diffuse sharply demarcated streptococcal infection of superficial lymphatic vessels.

Site

- More commonly affect the face.

Etiology

Organism

- Usually *strept pyogenes* [☑].

Predisposing Factors & Route

- See before

- **Etiology:** strept. pyogenes
- **C/P:** as before + rose pink + well-defined edge
- **Comp.:** general, elephantiasis, cavernous sinus thrombosis
- **Invest.:** CBC, C&S
- **TTT:** as cellulitis + isolation

Clinical picture

- Symptoms of toxemia are present.
- Locally the picture is similar to cellulitis but with **some differences**:
 1. The skin is rose pink.
 2. The edge is well defined, slightly raised and often shows minute vesicles just beyond the spreading margin.
 3. There may be islets of inflammation beyond the spreading margin separated from the main area by apparently normal skin.



Complications

1. Facial erysipelas may lead to cavernous sinus thrombosis.
2. Septicemia.
3. Recurrent erysipelas may block the lymphatics leading to elephantiasis.

Investigations

- Swabbing for culture and sensitivity.

Treatment

- As cellulitis + isolation of the patient as the disease is highly contagious.

Key points

Non-suppurative and sharply localized.

Boil (Furuncle)

Definition

- Acute staphylococcal infection of **hair follicles** → perifolliculitis (deep infection).

Etiology

Organism

- Usually Staph aureus → produce necrotoxin, responsible for the necrosis & sloughing.

- **Etiology**: staph. aureus + DM
- **C/P**: FAHM, pain, swelling, LN
- **Comp.**: as abscess + CS thrombosis
- **Invest.**: blood sugar for DM
- **TTT**: AAA + cause + comp.

Predisposing Factors & Route

- See before.

Pathology

Sites

- Hairy areas (face, neck, axilla, trunk, buttock).

Pathogenesis

- (Perifolliculitis – central necrosis & suppuration) → area around → red, hot, tender, a hair is coming out from it.

Clinical picture

Symptoms

- See before.

Signs

⇒ **General**:

- FAHM, marked constitutional symptoms are common



⇒ **Local:**

- Swelling → Hot & red, tender → at first indurated then soften at center → after 2-3 days → pus points & rupture.
- LNs → enlarged, elastic, tender, mobile.

Complications

- General+ Chronicity & Spread
- **Cavernous sinus thrombosis:** if manipulated or squeezed in the dangerous area of the face (the upper lip, septum of nose & adjacent area) → spread via pterygoid plexus or ophthalmic veins.
- Blind boil → Boil subsides leaving a thick indurated area.

Investigations

- Blood sugar for recurrent and severe cases.

Treatment

- See before (AAA + rest + hot fomentations) +
- Improve general condition e.g. vitamins, control DM, good nutrition.
- Antibiotic of choice are penicillinase resistant antibiotic as flucloxacillin (systemic antibiotics are often required).

Key points

1. Infection around hair follicle.
2. Complications: cavernous sinus thrombosis and blind boil.
3. Treatment: blood sugar control + improve general condition.

Carbuncle

(Infective gangrene of the nape) ✓

Definition

- Acute **staphylococcal** infection of S.C. tissues → Infective gangrene

Etiology**Organism**

- Usually Staph aureus → produce potent necrotoxin responsible for the necrosis of tissues & necrosis of blood vessels due to sloughing.

Predisposing Factors & Route

- See before
- **Diabetics are more common.**

Pathology**Sites**

- Nape of neck (commonest), back, face & dorsum of hand.

Pathogenesis

- Infection starting in hair follicles in skin.
- Then, Spreads to underlying fatty SC tissue → separate loculated abscesses → each abscess burst on the surface by a separate opening (i.e. a single carbuncle has multiple openings).

- **Etiology:** staph. aureus + DM
- **C/P:** FAHM, pain, swelling, LN
- **Comp.:** as abscess + CS thrombosis
- **Invest.:** blood sugar for DM
- **TTT:** AAA + cause + comp. + debridement

Clinical Picture

- ▣ There is usually severe toxemia.
- ▣ Usually starts as painful induration of the skin and subcutaneous tissue.
- ▣ The skin is red.
- ▣ As the swelling enlarges its central part become soft but usually no fluctuation can be detected.
- ▣ Multiple areas of skin thin out and separate forming multiple sinuses through which multiple sloughs separate slowly.
- ▣ Usually no frank pus forms.



Complications

- General + Chronicity & Spread
- Cavernous sinus thrombosis: if manipulated or squeezed in the dangerous area of the face (the upper lip, septum of nose & adjacent area) → Spread via pterygoid plexus or ophthalmic veins.

Investigations

- Blood sugar for recurrent or severe cases.

Treatment

- See before (AAA + rest + hot fomentations) +
- Antibiotic of choice is penicillinase resistant as flucloxacillin.
- Improve general condition e.g. vitamins, control DM, good nutrition.
- Once pus is formed → cruciate incision opening of all pockets.
- Debridement with removal of all the necrotic tissue.
- Dressing until healthy tissue is formed (culture is -ve).
- Leave to heal or use skin graft.
- Glycerine Mg sulfate may be applied until sloughing occurs.

Hidradenitis suppurativa

- ▣ Mixed staph. & strept. infection of apocrine sweat gland in perineum or axilla producing multiple abscesses & pus discharging sinuses.
- ▣ Infection is difficult to eradicate & can progress to chronicity with considerable reaction in perineum.
- ▣ **D.D.:**
 - Multiple anal fistulae (differentiated by absence of internal openings).
- ▣ **Treatment:**
 - Drainage of abscess followed by careful hygiene, painting with disinfectants & antifungal
 - Or excision of apocrine sweat glands followed by skin graft.

Key points

1. Infection starts in the hair follicle.
2. Commonest site is nape of the neck.
3. May be complicated by cavernous sinus thrombosis if manipulated or squeezed in dangerous area of the face.
4. How to drain.

Acute Lymphangitis

Definition

- It is infection of lymph vessels by organisms usually streptococci

Route of infection

- Entering through an abrasion or a wound in area drained by lymphatics

Clinical presentation

- Main trunk affection → red streaks in the overlying skin (tubular lymphangitis).
- Fever & rigors usually present

Treatment

- Systemic antibiotics.

Acute Lymphadenitis

Definition

- It is enlarged infected lymph nodes.

Route of infection

- Septic focus in drainage area

Clinical presentation

Symptoms

➔ **General** : FAHM

➔ **Local**

- Pain & swelling.

Signs of toxemia (e.g. fever & tachycardia)

Treatment

- Systemic antibiotics + drainage of septic focus.

Ludwig's Angina (Deep Cellulitis of the Neck)

➤ It is a surgical emergency.

Definition

- Bilateral diffuse cellulitis of the floor of the mouth & pharynx.
- Floor of the mouth is a CT-space divided by mylohyoid muscle into submandibular and sublingual spaces.**

➤ **Etiology**: strept.

➤ **C/P**: FAHM, swelling, dyspnea, dysphagia

➤ **TTT**: AAA + submental incision

Etiology

Causative organism

- Mainly mouth commensals (strept).

Route of infection

- Direct → from teeth (dental caries)
→ Ulcer of the tongue.

Clinical Picture

Symptoms

▪ General:

- Fever, anorexia, headache, malaise

▪ Local:

- Severe dysphagia and severe dyspnea as the tongue is pushed upward and backward → may obstruct food and air passages.
- Suffocation due to laryngeal edema or obstruction by the tongue.



Signs

▪ General:

- Fever.

▪ Local:

- Pharyngeal: edema of the floor of the mouth, the tongue is pushed upward and backwards.
- Cervical: swelling in submandibular region.

Treatment

▪ Medical:

- Massive doses of antibiotics. (early by penicillin)
- Bed rest in semi-sitting position to avoid airway obstruction.

▪ Surgical:

- Submental curved incision including the skin & deep fascia (there is no or little pus because suppuration seldom occurs).
- Tracheostomy when necessary.



Cellulitis of the neck

1. Superficial cellulitis: common & treated as cellulitis anywhere else, rarely if above the level of the hyoid bone can lead to glottic edema & asphyxia in children.
2. Deep cellulitis (Ludwig's angina)

Key points

1. Infection of the floor of the mouth.
2. Clinical picture of dyspnea & dysphagia + pharyngeal & cervical examination.
3. Sub-mental release incision including the skin and deep fascia.
4. Tracheostomy may be indicated.

Impetigo

Definition

- Superficial skin infection.

Etiology

Causative Organism

- Staph or strept or both.

Predisposing Factors

- Usually affects children (highly infectious).

Clinical Picture

1. Non-bullous type: blisters that rupture and coalesce to become covered with honey colored crust (commonly affects face around nose and mouth).
2. Bullous type.
3. Circinate type.
4. Ulcerative type: commonly affects lower limb.
5. Crusted type: usually affects the scalp.

N.B. Severe cases are associated with fever and regional lymphadenitis.

Complications

- Post-streptococcal glomerulonephritis.
- Scarlet fever.
- Erythema multiforme.

Treatment

- Topical neomycin or bacitracin after antiseptic lotion.
- Systemic antibiotic if fever or lymphadenitis or wide spread infection.

Necrotizing Fasciitis

Definition

- Diffuse non-suppurative spreading inflammation & infection extending to deep fascia.

Etiology

1. Following surgical procedures, e.g. drainage of peri-anal or ischiorectal abscess.
2. Following I.M injection or I.V infusion.

- **Etiology:** mixed (mainly strept.)
- **C/P:** FAHM, pain, swelling, crepitus, discharge, gangrene, MOF
- **Invest.:** CT/MRI, x-ray, lab. tests
- **TTT:** AAA + debridement + organ support

Causative organism

- The most common organism is **group A streptococcus**.
- Synergism (staph + strept + E.coli + anaerobes e.g. peptostreptococci...).
- Associations:
 - Meleny's synergism hospital infection. "Abdominal wall"
 - Fornier's gangrene of scrotum.

Predisposing factors

- Debilitating diseases, e.g. **DM**.
- Immunosuppressive drugs (usually affects immunocompromised patient)
- Malignant neoplasm.

Pathology

- Infection spreads along fascial planes & results in infectious thrombosis of blood vessels (skin & circulation) → superficial skin necrosis → hemorrhagic bulla is the 1st sign of skin death.

Clinical Picture

- ➔ The patient is usually moderately or severely toxic with lack of general symptoms
- ➔ The patient is alert & fully conscious.

Symptoms

- **General:** FAHM.
- **Local:** pain & swelling at the site.

Signs

- **General:** marked toxemia → fever & tachycardia.
- **Local:**
 - Edema beyond the area of erythema.
 - Crepitus.
 - Skin blistering.
 - Fever (often absent).
 - Greysih drainage (dish water pus).
 - Pink / orange skin staining.
 - Focal skin gangrene.
 - Finally → shock, coagulopathy & MOF.

▪ The most important signs are:

- Tissue necrosis
- Offensive discharge
- Severe pain
- Gas production

DD

- Cellulitis.
- Gas gangrene.
- Toxic shock syndrome.

Investigations

Laboratory: CBC, electrolytes, glucose, BUN, ABG & urine analysis.

Radiological:

- X-ray (demonstrate gas in tissue plains),
- CT & MRI to determine the presence of necrosis & surgical debridement.

Prevention

- Adequate debridement of the wounds.
- Blood loss replacement.
- Antibiotics for contaminated wounds specially in the postoperative.

Treatment

- Blood transfusion with nutritional support
- Antibiotic combination (Penicillin, Vancomycin & Clindamycin).
- Surgical debridement → followed by skin flap or graft.
- Amputations in selected cases.
- ICU admission in multi-organ failure.

Hand Infections

Classification of hand infection

1- Cutaneous & subcutaneous infections:

- Paronychia.
- Subcuticular + S.c. whitlow.
- Pulp space infection.
- Web space infection.

2- Facial space infection:

- Midpalmar space infection.
- Thenar space infection.
- Hypothenar space infection.
- Space of parona infection.

3- Synovial sheath infection:

- Acute digital tenosynovitis.
- Ulnar bursitis.
- Radial bursitis.

4- Bone & joint infections.

General

Etiology

Organism

- Staph (80%), Strept, E. coli, G -ve bacilli.

Predisposing Factors

- 1- Bad hygiene.
- 2- Bad general condition.
- 3- More in manual workers & housewives.

Route of Infection

- Usually **direct** spread of infection.
- Blood born infection is rare.

Clinical Picture

Symptoms

- **General** → FAHM.

- **Local:**

Pain → **dull aching** then become **throbbing** when pus is formed.
→ Decreased by elevation of the hand.

Swelling → according to site e.g. pulp space → distal phalanx, acute paronychia → nail fold, edema at the dorsum of the hand is common, irrespective of the site of infection.

Disturbance of function → limited movement.

Sings

- **General:** fever, tachycardia
- **Local:** hotness, redness, diffuse edema maximum on the dorsum of hand (loose dorsal skin) + LNs (enlarged elastic, tender and mobile) (axillary).

Complications

- **General:** toxemia, bacteremia etc...
- **Local:** according to type of infection e.g. pulp space infection → thrombosis of digital artery, acute paronychia → subungual abscess.

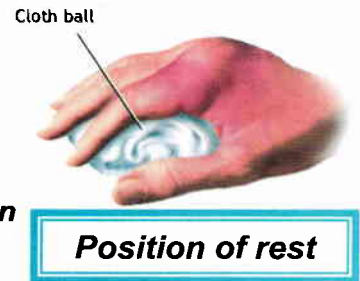
Investigations

- **X-ray:** if foreign body is suspected.
- **Blood sugar:** in recurrent cases may detect DM.

Treatment

Before Suppuration

- **General:** antibiotics, analgesics, antipyretic. (AAA)
- **Local:**
 1. **Hot fomentation.**
 2. **Arm position** → to diminish pain & edema.
 - Minor infection → arm to neck sling.
 - Major infection → elevated above level of body.
 3. **Hand position:**
 - If resolution is expected → **position of rest:**
 - ⇒ Description of position: the patient grasps a ball of cotton with max Flexion of little finger, least flexion of index with the thumb in opposition.
 - If stiffness is expected → **position of function**
 - ⇒ The fingers are approximated to the thumb as if holding something.
 - ⇒ Especially in tenosynovitis for fear of fibrosis of tendon & shortening → clawing of hand.



After suppuration → Incision & drainage

1. **Should be done once pus is formed.**
2. **Under general anesthesia**, ring anesthesia for minor cases (better to be avoided).
3. **Bloodless field by:**
 - Elevated the limb for 2 minutes.
 - Sphygmomanometer calf is inflated for 40 mmHg above systole.
4. **Incision.....**at the site of selection e.g. acute paronychia → oblique incision at the angle of nail fold and U-shaped incision if pus present all around.
 - **Never cross a skin creases** → will cause contracture.
 - **If in the digit → avoid mid line incision because** it will be painful, liable for trauma, ↓ tactile sensation
5. **All pus is removed** & granulation tissues are currated.
6. **No drain is allowed** because it causes pressure to delicate structures **but a piece of tulle-grass** might be allowed.
7. **Dry dressing is employed;** changed after the 1st day, then every 2 hours.

Special Types of Hand Infection

Acute Paronychia 30%

Definition

- Infection of the tissue surrounding the nail bed.

Etiology

Organism

Predisposing Factors & Route

- **Commonest** hand infection (30% of cases).
- Thorn is driven under nail.

Clinical Picture

Symptoms

- See before + swelling of nail fold.

Signs

- ⇒ **General:** see before.
- ⇒ **Local:**
 - See before +
 - Nail bed indurated.
 - Later on become cystic, yellowish.
 - At first at one side latter on U shaped around the nail fold.



Complications

- Trimming skin around nail Spread of infection under nail → subungual abscess.



Treatment

- See before +

Incision

- Oblique incision at the angle of the nail fold ± excision of the outer ¼ of nail.
- U – Shaped incision → if pus present all around the nail.
- Excision of outer quarter of nail → if pus under the nail (subungual abscess).

Acute subungual infection

- Infection of the space between subungual epithelium & their periosteum usually due to prick beneath the nail.
- **C/P:** severe pain & little swelling. The maximum tenderness is beneath the free edge of the nail where pus comes out.
- **ITT:** removal of small V from the center of the free edge of the nail.



Key points

1. Commonest hand infection (30%).
2. May be complicated by subungual abscess.
3. Specific clinical picture.
4. Comp.: subungual abscess
5. Oblique incision.

Chronic paronychia

Etiology

- Fungal infection (not related to acute paronychia).
- Patient whose hand is frequently emerged in water.

Clinical picture

- Itching and whitish nail bed.

Investigations

- Culture on sabouraud's agar.

Treatment

- Keep the hand dry + topical antifungal cream.
- If this fails, the nail fold is laid open & nail extraction may be required.

Pulp Space Infection (Felon)

- It is a surgical emergency.

Anatomy

- It is the SC compartment which is related to the palmer surface of the distal phalanx.
- It contains:
 - Fat.
 - Fibrous tissue septae (pus is trapped between it), which extend from the skin to periostium.
 - Digital artery which gives epiphyseal branch before it enters the space
- It is separated from the distal volar space by a deep fascia.

Definition

- Infection in SC compartment related to the palmer surface of the distal phalanx.

Incidence

- The 2nd common hand infection.

Etiology

- Usually a prick

Organism

- See before.

Predisposing Factors & Route

- See before.

Pathology

- See before.

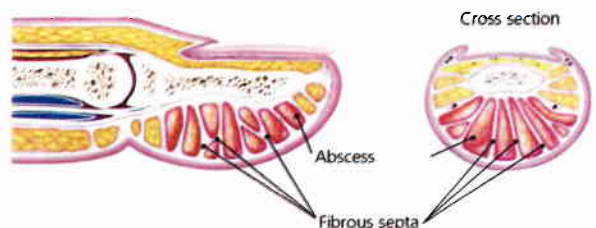
Clinical Picture

Symptoms

- See before + swelling of distal phalanges.

Signs

- ⇒ **General:** See before.
- ⇒ **Local**
 - See before +
 - The distal phalanx is first indurated.
 - Later on become cystic, yellowish.



Complications

- Thrombophlebitis of digital vessels → necrosis → infection (Osteomyelitis of the distal phalanx) → except epiphysis → Parrot peak deformity.
- Septic arthritis.

Treatment

- See before +

Incision

- Anterolateral on the lateral side of the distal 2/3 of distal phalanx or point of maximum tenderness.
- Counter incision → in severe cases.
- Hookey stick → for sequestrectomy
(The incision should be deepened to divide all septa).



Key points:

1. Anatomy of pulp space.
2. It is the 2nd common hand infection.
3. Specific clinical picture.
4. May be complicated by parrot peak deformity and septic arthritis.
5. How to incise.

Suppurative Tenosynovitis

1. Tenosynovitis of Middle 3 Fingers

Anatomy

- Each finger of these has a separate synovial sheath.
- **Proximal end:**
 - At the level of metacarpophalangeal joint.
- **Distal end:** at base of distal phalanges.
- They enclose the flexor tendons.
- Dilated Cul- de sac.

Etiology

Organism

- See before.

Predisposing Factors & Route

- See before.

Clinical Picture

Symptoms

- See before + swelling all around the finger.

Signs

- **General:** see before.

- **Local:**

- See before + classic signs described by Kanaval.
- 2. The affected finger is semiflexed with limitation of movements → hook sign.
- 3. Symmetrical swelling of entire finger.
- 4. Tenderness over the infected sheath specially its proximal "cul de sac" point



Complications

- Sloughing of the tendon due to interfering with nutrition of the tendon.
- Adhesions inside the synovial sheath → limitation of movement.
- Osteomyelitis - Arthritis.

Treatment

- See before +

Incision

- Transverse over proximal cul de sac → the sheath bulges & is incised → introduce a ureteric catheter → irrigate with penicillin.
- In severe cases → counter incision distally.

Key points:

1. Anatomy of synovial sheath of middle three fingers.
2. Specific clinical picture.
3. Hook sign.
4. How to incise.

2. Ulnar Bursitis

Anatomy

- Larger than radial bursa.
- Distally connected with synovial sheath of little finger.
- Proximally → run between the flexor retinaculum and pronator quadratus → extend to the forearm (**called space of Parona**).
- Envelop the flexor tendons of the medial 4 fingers.

Etiology

Organism

- See before.

Predisposing Factors & Route

- See before.

Clinical Picture

Symptoms

- See before + swelling of little finger, palm, distal part of forearm.

Signs

General:

- See Before.

Local:

- See before.
- Slight semiflexion of the little finger (hook sign).
- Limitation of movements of the medial 4 fingers.
- Edema of the dorsum.
- **Kanavel's sign**: tenderness over an infected ulnar bursa between transverse palmar creases & hypothenar muscles.

Complications

- Sloughing of the tendon due to interfering with nutrition of the tendon.
- Adhesions inside the synovial sheath → limitation of movement.
- Osteomyelitis.
- Arthritis.

Treatment

- See before +

Incision

- Along radial border of hypothenar eminence
- In severe cases → counter incision at the lower part of forearm

Key points:

1. Anatomy of ulnar bursa.
2. Specific clinical picture.
3. How to incise.

3. Radial Bursitis

Anatomy

- Small in size.
- Distally connected with synovial sheath of the thumb.
- Proximally → run between the flexor retinaculum and pronator quadratus → extend to the forearm (**called space of Parona**).
- Envelops the tendon of flexor pollicis longus.

Etiology

Organism

- See before.

Predisposing Factors & Route

- See before +

Clinical Picture

Symptoms

- See before + swelling of thumb, thenar eminence & distal part of forearm.

Signs

- **General:** see before.
- **Local:**
 - See before + thumb is semiflexed.

Complications

- Sloughing of the tendon due to interfering with nutrition of the tendon.
- Adhesions inside the synovial sheath → limitation of movement.
- Osteomyelitis
- Arthritis.

Treatment

- See before +

Incision

- On the ulnar side of thenar eminence stopping proximally 1.5 inches distal to the distal crease of the wrist to avoid injury of the motor branch of median nerve
- In severe cases → counter incision

Key points:

1. Anatomy of the radial bursa.
2. Specific clinical picture.
3. How to incise.

Web Space Infection

Web Space Infection

Anatomy

- Triangular region between the dorsum & ventral skin present at the bases of the fingers.
- They contains: fat, digital vessels & nerves, lumbricals & Interossei.
- 1st & 2nd web spaces are connected to thenar space, while 3rd & 4th web spaces are connected to palmar space.

Etiology

Organism

- See before.

Predisposing factors & Route

- See before +
- **Extension from:**
 - Adjacent web spaces.
 - Deep mid palmar space.

Clinical Picture

Symptoms

- See before + swelling of the web space.

Signs

- **General:**
 - See before.
- **Local:**
 - See before +
 - Swelling of the web space → Hot & red, Tender.
 - Sign of victory (the finger can not be approximated)

Complications

- See before +
- **Spread to** deep mid palmar space & adjacent web space.

Treatment

See before +

Incision

- A transverse incision over the web one cm from the free margin.
- Counter incision posteriorly → severe cases



Key points:

1. Anatomy of web space.
2. Specific clinical picture.
3. How to incise.

Thenar Space Infection

Anatomy

- Ant → thenar muscle, radial bursa, and flexor pollicis longus.
- Post → adductor pollicis.
- Med → deep mid palmar space.
- Distal → extend to the web of the thumb.

Etiology

Organism

- See before.

Predisposing factors & Route

- See before.
- **Extension from:**
 - Tenosynovitis of the thumb.
 - Deep mid palmar space.

Clinical Picture

Symptoms

- See before + swelling of the thenar eminence

Signs

- **General:** see before
- **Local:**
 - See before +
 - Swelling of the thenar eminence.
 - Edema of the dorsum of the hand (marked).

Complications

- See before + **Spread to** deep mid palmar space & adjacent web space.

Treatment

- See before +

Incision

- Drained through an incision at the site of maximal tenderness or where pus points at the skin.
- An incision is done along the medial side of the thenar eminence should stop 2 cm distal to the distal wrist crease to avoid injury of the motor branch of medial nerve.

Key points:

1. Anatomy of thenar space.
2. How to incise.

Superficial Mid Palmar Space Infection

Anatomy

- Anterior → palmar aponeurosis.
- Posterior → flexor tendons.

Etiology

Organism

See before

Predisposing factors & Route

See before

Clinical Picture

Symptoms

See before

Signs

General

- See before.

Local

- See before +
- Obliteration of the palm concavity (frog hand).
- Edema of the dorsum of the hand.

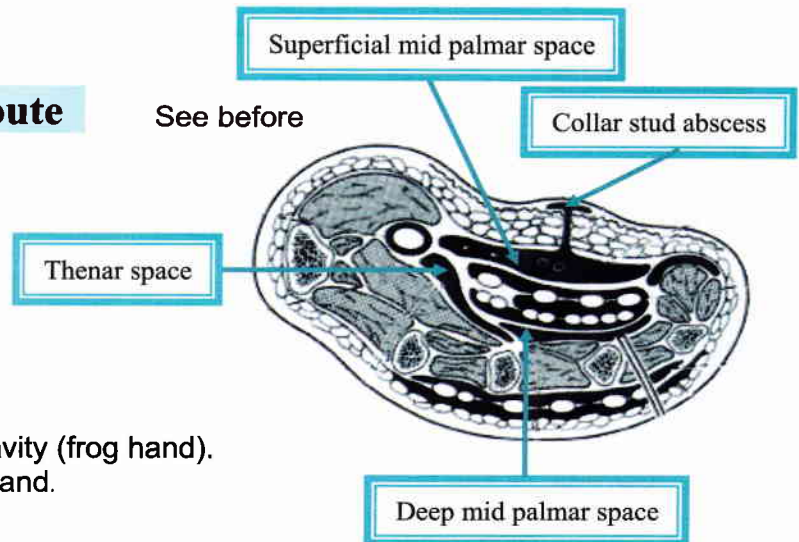
Complications

- See before +
- Formation of Collar stud abscess:
 - Locule lies in the SC tissue → subcuticular whitlow.
 - Locule in the superficial mid palmar space.
 - Hole in the palmar aponeurosis connecting them.

Treatment

Incision

- A transverse incision at the line of the crease passing over the site of maximum tenderness.
- The palmar fascia is divided in longitudinal direction to avoid injury to digital N. & V. (Hilton's method).



Key points:

1. Anatomy of mid-palmar space.
2. Specific clinical picture.
3. May be complicated by collar stud abscess.
4. How to incise.

Deep mid palmar Space Infection

Anatomy

- Ant → flexor tendon of the medial 3 fingers.
- Post → fascia covers the interossei.
- Lat → fibrous band from palmar apponeurosis to 3rd metacarpal.
- Medially → fibrous band from palmar apponeurosis to 5th metacarpal.

Etiology

Organism

- See before.

Predisposing factors & Route

- See before.

Clinical Picture

Symptoms

- See before

Signs

- **General** → see before
- **Local** → see before +
 - Obliteration of the concavity of the palm (frog hand).
 - Edema of the dorsum of the hand.

Complications

- See before + - Web space infection of the medial 3 webs - Thenar space infection

Treatment

- see before +

Incision

- Transverse incision over the space
- Counter incision from the web space → if web space is affected
- Semiflexion of the medial 3 fingers

Key points:

1. Anatomy of deep mid-palmar space.
2. Specific clinical picture.
3. How to incise.

Subcuticular Whitlow

- It is pus under the cuticle within the epidermis.
- It may be communicated with deeper abscess (collar stud abscess).

Treatment

- The raised epidermis is incised
- Gentle probing is done for a track extending to a deeper abscess if present.

Ingrown Toe-Nail (Onychocryptosis)

Definition

- Common problem in which a sharp edge of the big toe nail impinges on the adjacent skin fold leading to necrosis of the nail sulcus due to persistent contact of the nail edge.

- **Incid.:** big toe of young ♂
- **Etiology:** wrong trimming, bad(shoes & nails)
- **Comp.:** suppuration
- **TTT:** conservative or excision

Incidence

- Young males predominate

Site

- It occurs more in the big toe.

Etiology

1. Faulty nail trimming → oblique trimming of the nail sides may leave behind a sharp spike that starts the condition.
2. Wearing tight shoes → thrusts this spike in the soft tissues
3. Nail abnormalities as hypercurved nail.

Complication

- This leads to infection & suppuration in the nail fold → may be made worse to cut away the nail.

Treatment

Conservative (in early cases)

1. A pledget of gauze soaked in an antiseptic is inserted beneath the ingrowing part of the nail to raise it up and to ease away the injured tissues → The pledget is changed daily until the nail grows past the nail fold.
2. Correct trimming of the nail (square nail trimming where the centre of the nail is kept at the same level or even shorter than its edges).
3. Avoid tight shoes.
4. Keep the foot clean & dry.

Operative

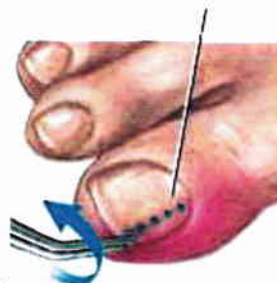
➤ Indications:

1. Failure of conservative treatment.
2. Suppurative cases

➤ Technique:

1. Wedge (segmental) excision of the germinal matrix is the definitive treatment.
2. Excision of the nail with periosteum.
3. If the area is heavily infected, the wound is left open to granulate, otherwise its edges are loosely approximated.

Excision line



Nail portion removed



Prognosis

- Recurrence is common with regrowth of the nail.

Inflammation of the oral cavity (Stomatitis)

- This is a group of inflammatory, erosive & ulcerative conditions that affect the mucous membrane of the oral cavity.

Predisposing factors

1. Anemia & Vit. B12 deficiency → thin atrophic epithelium & loss of tongue papillae.
2. Immunodeficiency & AIDs.

Types

A. Aphthous stomatitis

- Painful ulcers up to 0.5 cm across.
- **Shape:** rounded or oval.
- **Base:** yellow.
- **Margin:** red.
- They heal normally within 10 – 14 days.

B. Herpes simplex infections

- Small vesicles → rapidly break down → small, yellow ulcers with bright red margins.
- **Site:**
 - Gingiva, cheeks, lips & tongue.
 - Skin of the cheek & around the nostrils.
- **Clinical picture:** fever & enlarged submandibular L.Ns.

C. Monilial stomatitis

- **Definition:** a fungal infection by Candida species characterized by formation of white membranous lesions.
- **P.D.F.:** chronically ill patient (under cytotoxic drugs, cortisone therapy or prolonged treatment by broad spectrum antibiotics).
- **Site:** tongue.
- **Treatment:** gentian violet & amphotericin B lozenges.

Inflammation of the tongue (glossitis)

- Minor degrees of glossitis can occur due to scalding fluids, sepsis, diabetes, & alcoholism.
- This form presented by a painful red glazed tongue.

I- Chronic superficial glossitis

Etiology

- Due to chronic irritation (smoking, sharp tooth, syphilis, & spices).
- Incidence
- Middle & old ages.

Pathology

- **Site:** anterior 2/3 of the tongue.
- Hypertrophied papillae, leukoplakia, red glazed tongue, cracks, fissures & warty projections.
- Hypertrophy of the papillae → red hyperemic patches.
- Epithelial overgrowth → thickened indurated & opaque epithelium.
- So, the red patches are replaced by white ones → constituting **leukoplakia**.

- The epithelium may shed over a considerable area → red glazed tongue.
- Submucous fibrosis occurs → ulcers, fissures & warty projections.

Prognosis

- It is precancerous.

Treatment

1. Elimination of the irritating source.
2. Mouthwash.
3. In resistant cases → excision of the lesion.

II-Tuberculous glossitis

Etiology

- Due to autoinoculation from pulmonary tuberculosis, through the septum.

Pathology

- Either a circumscribed interstitial tuberculoma or as a shallow oval painful ulcers with overhanging margin.

III-Syphilitic glossitis

- The tongue may be affected in any of the **3 stages of syphilis**:

1- Primary stage:

- An extragenital chancre at the tip of the tongue.
- It is painless.
- There is great enlargement of the submandibular L.N.

2- Secondary stage:

- Multiple shallow snail track ulcers appear on the sides & undersurface of the tongue.

3- Tertiary stage:

- May take the form of a gumma as chronic superficial glossitis.
- A gumma presents as a slowly growing midline swelling.
- The overlying tissues get necrosed & separate as wash leather slough → leaving a deep crater like ulcer.
- This has to be differentiated from carcinoma which usually doesn't occur in the midline & which produces tethering of the tongue.

Alveolar abscess

- It may result from the spread of infection from necrotic pulp into the periapical tissue.
- Pus may burst under the periosteum → form a subperiosteal abscess → points externally or in the maxillary antrum.
- Infection may spread to the cavernous sinus.

Clinical picture

1. Marked toxemia.
2. Severe pain & cheek swelling with gum inflammation.
3. Enlarged tender regional L.N.s

Treatment

1. Antibiotics.
2. Extraction of the offending tooth to provide drainage.
3. Incision and drainage of the subperiosteal abscess.

	Type of incision
Acute paronychia	- Oblique incision at the angle of the nail fold - U – Shaped incision
Pulp space infection	- At point of maximum tenderness - Or anterolateral on the lateral side of the distal 2/3 of distal phalanx - Counter incision → in severe cases.
Web space infection	- A transverse incision over the web one cm from the free margin. - Counter incision posteriorly → severe cases
Thenar space infection	Curved incision done along the first web space
Superficial mid palmar space infection	A transverse incision at the line of the crease
Deep mid palmar space infection	- Transverse incision over the space - Counter incision from the web space → if web space is affected
Tenosynovitis of middle 3 fingers	- Transverse over proximal cul de sac - introduce a ureteric catheter → irrigate with penicillin
Ulnar bursitis	- Along radial border of hypothenar eminence - In severe cases → counter incision at the lower part of forearm
Radial bursitis	- On the ulnar side of thenar eminence - Severe cases → counter incision



More Details about Bacteriology

Staphylococci

- Gram +ve cocci.
- Most important pathogen is staph aureus (coagulase +ve) found in nasopharynx up to 15% of population.
- Other strains:
 - MRSA can cause epidemic hospital acquired infections some of its strain are now also resistant to vancomycin.
 - Staphylococcus epidermidis also called coagulase –ve staphylococci (CNS) represent a major threat in vascular and orthopedic surgery.
- Most hospital S.A. are now B-lactamase producer and are resistant to penicillin but most strains (MRSA) remain sensitive to flucloxacillin, vancomycin and some cephalosporins.

Streptococci

- Gram +ve forms chains.
- Most important is B-hemolytic strept found in pharynx of 5-10% of population.
- **Other strains:**
 - Group A strept also called strept pyogenes. It is the most pathogenic. It has the ability to spread causing cellulitis through release of streptolysin and streptokinase.
 - Strept fecalis: is an enterococci often found in synergy with other organisms as hemolytic strept.
 - Peptostreptococci is anaerobe.
- All strept remain sensitive to penicillin and erythromycin.

Aerobic Gram –ve bacilli

A. Escherichia Coli (E. coli):

- Found in the intestinal tract of all human beings.
- Many species, some are commensals while others are pathogenic.
- Responsible for most abdominal and urinary tract infections.

B. Klebsiella:

- Encapsulated gram –ve bacilli.
- Found in the respiratory tract.
- In debilitated patients, it may cause fatal pneumonia.

C. Proteus vulgaris:

- Common cause for urinary tract infection, wounds and burns (as a component of mixed infections)

D. Pseudomonas aeruginosa:

- Found in human feces in 20% of cases.
- Common as 2nd invader especially in wounds and burns.
- Recognized by blue-green color of the pus and its odor.

Anaerobic bacteria

- Obligatory anaerobes. The most important are Peptostreptococci and Bacteroides.
- May cause wound infection and even septicemia.
- Characterized by gas formation, tissue necrosis and foul-smelling discharge.

Tetanus

Definition

- Acute specific infection leading to ↑ nervous excitation due to release of neurotoxin.

Incidence

- Annual incidence: 0.5-1million case.
- Predominant disease of undeveloped countries.

Etiology

Causative organism

- **Clostridium tetani:** Gram +ve, anaerobic bacilli, spore forming drum stick appearance

Predisposing factors

- 1- ↑ organism content: (as in contaminated wounds occurring in fields and streets)
- 2- ↓ O₂ content:
 - a- From air:
 - Deep lacerated wounds.
 - Associated pyogenic infections.
 - b- From blood:
 - Hemorrhage and shock (general cause)
 - Lacerated devitalized wounds
 - Compartmental syndrome
- c- Lack of proper sterilization of catgut and instruments

} Local

Route of infection

- 1- Wounds: → especially occurred in fields or streets (contamination with animal faeces is common)
- 2- Post-operative tetanus: →lack of proper sterilization of cat gut.
- 3- Tetanus neonatorum: → infected instrument (septic delivery).
- 4- Infected umbilical stump.

Pathology

(Anaerobic infections usually do not spread by blood)

- The organism secretes an exotoxin (neurotoxin) which has two actions:
 - A. Anti-Choline esterase action: → Interferes with destruction of acetylcholine at the motor end plate →tonic rigidity of muscle.
 - B. Extreme excitability of the motor neurons (ant. horn cells): → Leading to convulsion attacks on exposure to minor stimuli.
- **The exotoxin reaches the CNS by one of the following routes :**
 - 1- **Blood spread:** this explain the generalized form of the disease.
 - 2- **Spread along motor neurons:** this explain the localized form of the disease.

Once the toxin had reached the CNS it becomes fixed & can't be neutralized by the antitoxin

- **Etiology:** Cl.tetani, ↑org., ↓O₂ content
- **C/P:** FAHM, pain, swelling, convulsions
- **Comp.:** asphyxia, HF, hyperpyrexia
- **Invest.:** smear, organ profile
- **TTT:** prophylactic &curative

Clinical picture

Incubation period

- Immunized → from 11 days to several weeks & months.
- Non-immunized → 1 – 15 days.

Symptoms

- A. General:** FAHM + the patient is alert, vague and mild symptoms as headache, fever, anorexia & rapid pulse.
- B. Local:**
- Pain → at site of wound.
 - Swelling → local small with minimal infection reaction.
 - **Convulsions:** the time between appearance of the 1st symptom and occurrence of the 1st reflex spasm is called period of onset (if < 48 hours → bad prognosis).

Signs

A. General:

1- Stage of tonic contraction (prodromal stage):

- **Trismus (earliest sign)** → spasm of masseter & temporalis muscles.
- Risus sardonicus → spasm of facial muscles → **bitter smile (painful smile)**
- **Opisthotonus** → Stiffness of neck & body arched backwards.
- Dysphagia → spasm of pharyngeal muscles.
- Dyspnea & stridor → spasm of laryngeal & respiratory muscles.

2- Stage of clonic contraction: (Reflex convulsion)

- Clonic spasm on top of tonic spastic muscles (i.e. **relaxation is incomplete between contractions**).
- Excited by light, noise, or any other external stimuli.
- **Muscles are spastic in between attacks** (violent & continuous).
- Profuse sweating.

B. Local (wound examination):

- Red, hot, tender & small (± infected with other organisms).

Complications & causes of death

Mortality rate 45%

1. Exhaustion (from severe convulsions).
2. Hyper-pyrexia (excessive energy production) only if convulsions.
3. Asphyxia (laryngeal spasm) → respiratory failure.
4. Heart failure.



DD

A- Colonic contraction: (no tonic phase between convulsions)

1. **Strychnine poisoning.**
2. **Hydro-phobia in rabies:** complete relaxation between convulsions.

B- Tonic contraction:

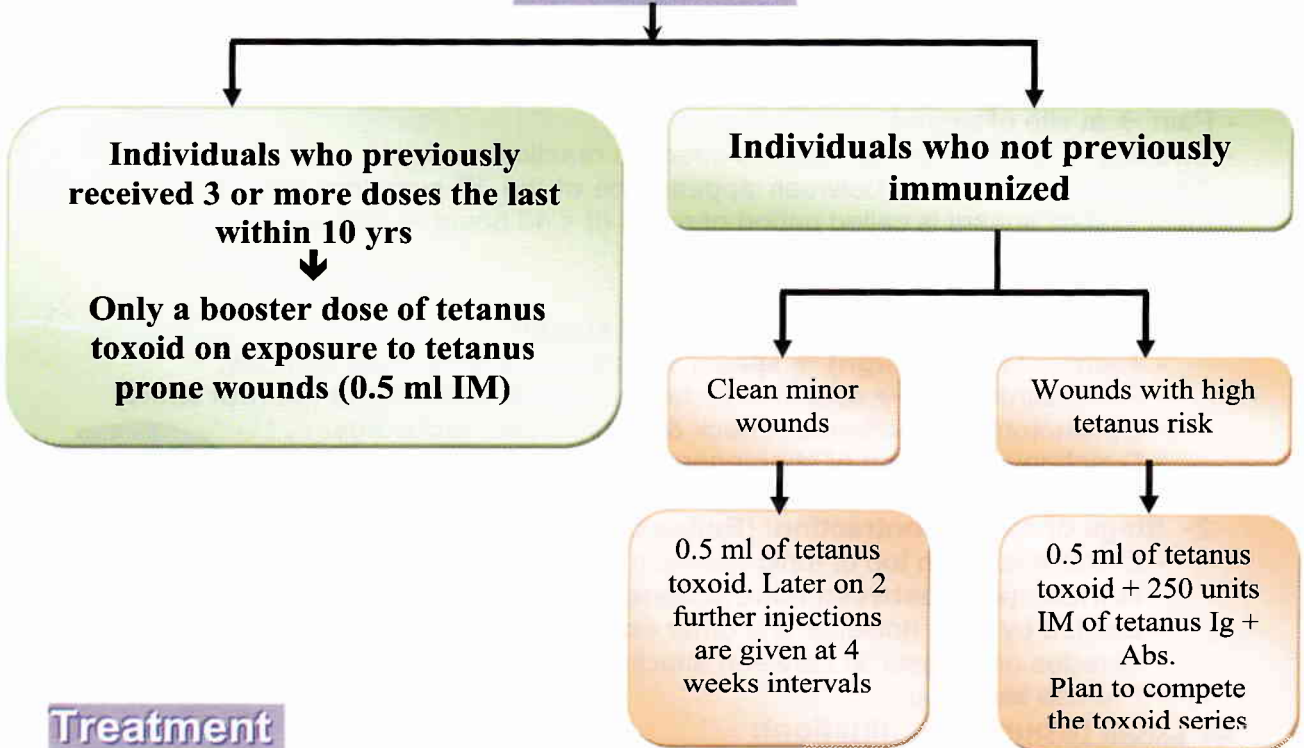
1. **Tetany** (carpopedal spasm).
2. **Trismus due to local causes** (painful).
3. **Meningitis** (severe toxemia + neurological signs).

Investigations

- ABGs.
- Organ profile.
- **CBC:** leukocytosis.

- **CSF:** normal.
- Smears from wound → Gm +ve spore forming organism with drumstick appearance.
- Spatula test is a bed side test done during stage of tonic contraction:
 - Simple: involve touching oropharynx with spatula (no adverse effects).
 - **If normal:** gag reflex.
 - **If tetanus:** reflex spasm of masseter & bite of spatula

Prevention



Treatment

1. Neutralize toxin with TIG. The dose in established cases is 3000-6000 units IM, given preferably in the proximal portion of the wound extremity or in the vicinity of the wound. Repeated doses may be needed.
2. Excise & debride the suspected wound under anaesthesia. the wound must be left open & may be treated with hydrogen peroxide.
3. The patient should be protected from sudden stimuli, unnecessary movements & excitement. Barbiturates are used cautiously as they often cause cardio-respiratory failure. curarization with mechanical ventilation is a better alternative to large cardiosuppressent doses of barbiturates.
4. The patients with respiratory problems may require tracheostomy. The patient should be intubated once respiratory problems appear.
5. Aqueous penicillin G, 10-40 million units a day.
6. The patient is isolated in a dark quiet room & nutrition is maintained by nasogastric tube. All doses should be modified in tetanus neonatorum.

Prognosis

- Death rate is 30-60% in established tetanus with respiratory insufficiency.
- Death rate is inversely proportionate to the length of the incubation period and directly proportionate to the severity of symptoms.
- One attack of tetanus does not confer a life-long immunity.
- Patients who recover from an attack need to have an active immunization schedule.

☞ Tetanus Ig should be taken at a different site & a different syringe from tetanus toxoid.

☞ **Tetanus toxoid:**

- Formaline attenuated vaccine given in three separate doses to give 5 years protection after which **10 years** booster dose confirms immunity.

☞ **Vaccination:**

- Every child should receive vaccination by DPT (Diphtheria, Pertussis, Tetanus) 2, 4, 6 m & booster at 18 m, by 5 yrs we give only DT.
- High risk group e.g. house workers, must be vaccinated every 5 yrs.

Key points:

1. Predisposing factors: contaminated wound in field or street.
2. Mechanism of action via neurotoxin (tonic then clonic convulsions).
3. Clinical picture and cause of death.
4. Proper prophylaxis by good wound care.
5. Value of toxoid and role of immunoglobulin and penicillin.
6. Ventilator may be needed in severe cases.

Gas Gangrene

Definition

- Acute specific infection leading to spreading of gangrene with excess gas formation.

Etiology

Causative organism

- **Gram +ve spore** forming anaerobic bacilli (that **secretes lethal exotoxin**).
- Two groups of organisms.

Saccharolytic group (act on CHO)

- **Cl. Welchii.**
- Cl. Oedematiens.

- Cl. Septium.

Proteolytic group: (act on protein)

- Cl. Sporogens.
- CL.Tertium.

- **Cl. Histolyticum.**

Predisposing factors

- 1- ↑ organism content: (as in contaminated wounds occurring in fields and streets)
- 2- ↓ O₂ content:
 - a- From air:
 - Deep lacerated wounds.
 - Associated pyogenic infections.
 - b- From blood:
 - Hemorrhage and shock (general cause)
 - Lacerated devitalized wounds
 - Compartmental syndrome
- c- Lack of proper sterilization of catgut and instruments.
- 3- Wounds: → especially occurred in fields or streets (contamination with animal feces is common)
- 4- Endogenous infection: may occur as in lower limb amputation or large bowel operation or septic abortion.

- **Etiology:** Cl.epecies, ↑org., ↓O₂ content
- **C/P:** as tetanus + crepitus(gas) + gangrene
- **Comp.:** severe toxemia, MOF
- **Invest.:** as tetanus + imaging
- **TTT:** prophylactic &curative

Pathology

(Anaerobic infections usually do not spread by blood)

A. Local:

- ➔ Due to proliferation of the organism & toxin diffuse in surrounding tissue destroying microcirculation → more proliferation.
- Saccharolytic group → act on glycogen of dead muscles → gases elevates sarcolemma → open the way for invasion with proteolytic group
- Proteolytic group → act on proteins of dead muscles → $\text{NH}_3 + \text{H}_2\text{S}$ → offensive odor, FeS → stain muscles black

B. General: (Due to exotoxin = alphatoxin)

- Hemolysis of RBCs → slight jaundice.
- Thrombosis of blood vessels → muscle necrosis.
- Severe toxemia and MOF e.g. liver and kidney failure.

Clinical picture

Incubation period

- Few hours – Few days

Symptoms

A. General

- FAHM (**Slight fever or even subnormal temp**).

B. Local

- Pain → at first marked but disappears later on.
- Swelling → **wound is black & edematous with foul odor with watery discharge.**
- Disturbance of function → paralysis of affected organ.

Pain and swelling at the site of a recent wound ± foul-odor discharge

Signs

A. General:

- Tachycardia: earliest sign.
- **Icteric Jaundice** → may appear from **absorbed toxins**, **anemia** from hemolysis of RBCs.
- MOF (multiple organ failure) → ARF, ARDS
- Shock: dry tongue, hypotension ...etc.

B. Local:

- (Signs of gangrene)
- The wound shows swelling, gas bubbles with **crepitus** sanguinous **watery discharge** with foul odour
 - The affected muscle shows
 - discoloration: red → green → black
 - No contraction if pinched
 - No bleeding if cut
 - the surrounding skin may shows
 - Discoloration green or black
 - Blebs or foul smelling dark fluid.
 - Pain and numbness in the affected area



Complications

(Causes of death)

- Severe toxemia and multi-organ failure.

DD

1. Infection with gas forming organism (e.g. Clostridial species, E.coli, Klebsiella species or Enterococci)
 - High fever
 - Excessive pus formation with no peripheral circulatory failure & no offensive discharge
 - Normal muscles
 - Leucocytosis.
2. Surgical emphysema: (Gas under the skin).
 - No toxemia.
 - The wound is healthy

Investigations

- **TLC:** Leucopenia & anemia.
- Raised serum bilirubin (hemolysis).
- Smear & culture from the discharge → gram +ve bacilli.
- Plain X- ray will show gases in the tissue planes.
- **CT – MRI:** for better evaluation of tissue planes.

Treatment

Prophylactic

Management of the wound:

- | | |
|---|--|
| a. Skin is incised | c. Muscle debridement |
| b. Deep fascia is opened | d. Wash the wound with H ₂ O ₂ |
| e. If less than 8 hrs → close the skin loosely without deep fascia. | |
| f. Avoid deep bandage | |
| g. If more than 8 hrs → the wound is left opened | |

A. General

- Sterile instruments & sutures.
- In nosocomial infection → hospital is closed & sterilized.

B. Specific

- Polyvalent anti-gas gangrene serum: 10 CC IM/d for 3 days (**doubtful value**).
- Antibiotics e.g. penicillin G.

Curative

Management of the wound is the most important initially in treatment

A. General

- R&M → see before +
- Isolate the patient in a separate place.
- Blood transfusion & IV fluids.
- Hyperbaric oxygen (at 3 atmospheric pressure for 1-2 h & repeated every 6-12 h) it inhibit bacterial invasion & toxin production but doesn't eliminate the focus of infection (controversial).

B. Specific

- **Anti-gas gangrene Ig:** 100 CC IV/d for 10 days (of doubtful value).
- Crystalline penicillin: 20 – 40 million U/day.

C. Others

- Massive type → high amputation above all affected muscles
- Localized type → excise the muscles affected + dusting the wound by penicillin powder + free drainage of the wound.

Prognosis

- **Mortality :** 20% it varies according to extent and severity if injury and timing of treatment
- **Limb salvage:** the rate of saving a functioning limbs not favorable , the myonecrosis occurring render some cases inevitable (life-saving amputation).

Chronic specific infections

Tuberculosis

Etiology

Causative organism:

- *Mycobacterium tuberculosis* which is Gram +ve, acid-fast bacilli of two types, human type and bovine type.

Predisposing factors:

- There is a recent rising incidence accompanied the increase of the incidence of (AIDS).
- The disease still common in poor underdeveloped countries

Mode of infection:

- inhalation of infected droplets or swallowing contaminated food especially milk.

Pathology

Microscopic picture:

The tubercle is formed of:

- a. Epithelial cells in the central part with the bacilli.
- b. Lymphocytes at the periphery.
- c. Langhan's giant cells with numerous nuclei arranged commonly in horse shoe pattern.
- d. Fibrous layer (outer most), may be extensive if the host resistance is high and may be calcified.
- e. Caseation: is a form of coagulative necrosis near the center of the tubercle, areas of Caseation may coalesce and lead to formation of cold abscess.

Spread

- The disease may spread in different ways:
 - a. Direct extension to adjacent structures.
 - b. Lymphatic spread to lymph nodes.
 - c. Hematogenous spread leading to distant secondary lesion or miliary tuberculosis.
 - d. Via natural passage as in the urinary tract and air passage.

Clinical picture

- A. **General:**(tuberculous toxemia) Loss of weight, Loss of appetite, Night fever and Night sweating.
- B. **Local** manifestations depend on the affected site, but could be in form of:
 - i. Cold abscess: soft fluctuating mass with minimal signs of acute inflammation unless become secondary infected.
 - ii. Tuberculous ulcer: irregular small ulcers with undermined cyanotic edges.
 - iii. Chronic sinuses: may be multiple and surrounded by ulcers.

Investigations

1. Bacteriological examination of the exudates and aspirated material by Z.N. stain and culture on specific media.
2. Polymerase chain reaction (PCR) is an accurate fast test.
3. Tuberculin skin test only useful when negative.
4. X rays of affected organs as lung and bones.
5. Histopathological examination.

Treatment

A. Chemotherapy

- Using antituberculous as:
 - INH (Iso-Nicotinic acid Hydrazide), rifampicin, pyrazinamide and ethambutol
 - ciprofloxacin, aminoglycosides & cycloserine if there is resistance to the classic line of treatment & it is better to do culture & sensitivity.
- At least two drugs have to be used at one time.
- Treatment has to be extending for 6 or 9 months.

B. Improving the general condition of the patient

- Using tonics, vitamins, good rich diet with high protein contents.

C. Local treatment

- Depends on the site and type of lesion.

	Actionmycosis	Nocardiosis
Etiology	Actinomycetes - Gram-positive - Strict anaerobes - Part of the normal flora of the human oropharynx & tonsils.	Nocardiae madurae - Gram-positive - Aerobes - Rarely found in the normal flora of the respiratory tract.
Pathology	- Sinuses discharging small yellowish sulphur granules  <u>Spread</u> - No lymphatic spread	The localized type: - Sinus discharging black granules. Systemic type. - Rare
Clinical picture	Faciocervical type (60%) Abdominal type (20%) - Mass in the RIF. - Liver abscess. Thoracic type (15%) Cutaneous type (5%) 	The localized type "Madura foot" (Mycetoma) belongs to this type. - Affects the extremities and infection occurs through abrasions in bare feet. - Extensive bone destruction but little systemic illness.
Treatment	Penicillin (5-20 million units daily) for many weeks or Tetracycline. Surgical excision	- Sulfonamides ± Minocycline - Surgical drainage ± Amputation

Non spore forming infection

↪ **Bacteriodes: B.Oralis and B.Fragilis.**

- Normal inhabitants of the respiratory system and the intestine.
- The infection is always endogenous.

↪ **Anaerobic streptococci:**

- Produces cellulitis or myositis.
- Progressive synergistic gangrene with other pyogenic organisms.

↪ **Actinomycosis:** see above.

Surgical Site Infection (SSI)

(Surgical Wound Infection)

Definition

- They are infections of tissue, organs & spaces exposed during the performance of invasive surgical procedures.
- **Incisionals** → they are either superficial → limited to skin & S.C. tissue
→ Deep in musculoaponeurotic.
- **Organs**
- **Spaces** → e.g. subphrenic or pelvis

- **Etiology:** single or multiple organism
P.F.=Bad (wound, patient, surgery)
- **C/P:** FAHM, pain, swelling, LN
- **Comp.:** as abscess + gangrene, SIRS, MOF
- **TTT:** -prophylactic: acc. to class
-curative: AAA + drainage

Classification

A. Clean wounds (class I):

- Elective non-traumatic wounds in which the gastrointestinal, urinary or respiratory tracts are not entered.
- Risk of SSI → less than 2%.

B. Clean contaminated (class II):

- Elective surgery into the gastrointestinal, urinary or respiratory tracts with no significant spillage.
- Risk of infection → 2-5%.

C. Contaminated wounds (class III):

- Open accidental wounds encountered within 4 hours.
- Gross spillage from GIT.
- Incision through non-inflamed, non-purulent tissues.
- Risk of infection → 10-20%.

D. Dirty wounds (class IV):

- Traumatic wounds more than 4 hours.
- Purulent infection or necrotizing soft tissue infection.
- Perforated viscus accompanied by high degree of contamination.
- Risk of infection → up to 40%.

Causative Organisms

- The most common causative organisms associated with wound infections include Staphylococcus aureus / MRSA, Streptococcus pyogenes, Enterococci and Pseudomonas aeruginosa
- Synergy of gram -ve bacilli and anaerobic bacteroids (abscess after abdominal surgery).

Source of Infection

- Endogenous: human body flora.
- Exogenous: from surgical team, instruments, dressing etc....

Predisposing Factors

✚ <u>General patient characteristics</u> (↑ affinity to opportunistic infection)	• Age, obesity, malnutrition • Metabolic disorders • Anemia • Malignant disease • Immunosuppression
✚ <u>Wound characteristics</u>	• Nonviable tissue in wound • Foreign bodies • Tissue ischemia • Hematoma formation
✚ <u>Operative characteristics</u>	• Nature of surgery (most important factor) • Poor surgical technique • Long operation time (>2 hours) • Intra-operative contamination • Prolonged preoperative stay • Presence of foreign bodies or prosthetic material.

Clinical Picture

Usually appears between the 5th & 10th day postoperative.

Symptoms

- General
 - Fever, anorexia, headache & malaise.
- Local:
 - Pain.
 - Swelling.
 - Disturbance of function: The wound hasn't healed within 10 days after the injury.

Signs

- General:
 - Fever & tachycardia
- Local:
 - The wound show: redness, tenderness, may ooze pus or cloudy fluid from the wound & yellow crust has formed on the wound.
 - LNs: large and tender.
 - Fluctuant areas and crepitus are occasionally felt



Complications of surgical infections

1. Spread of infection by several mechanisms

a. Direct

- i. Necrotizing infections
- ii. Abscesses
- iii. Phlegmons. These contain little pus but much oedema

b. Lymphatic spread: especially streptococcal infection

c. Blood stream spread

2. Fistulae and sinuses

3. Necrosis or gangrene of the affected part

4. Suppressed wound healing

5. Immunosuppression and superinfection

6. Systemic inflammatory response syndrome (SIRS) and Multiple organ system failure (MOSF): the most serious complication → See "Septic shock"

Investigations

Laboratory ▪ CBC: ↑TLC

Microbiology

- Swabs are commonly sent for culture. However, pus if available is a better sample than a swab.
- Other fluids or tissue biopsy samples may also be cultured.
- Blood culture is recommended in febrile patients

D.D.

- Other causes of wound swelling, e.g. hematoma.

Other causes of postoperative fever, e.g. DVT, chest infection etc....

Criteria for diagnosis

- Surgical site infections must fulfill the following criteria:
 - Infection must occur within 30 days of surgery.
 - Infection must involve only the skin and subcutaneous tissue.
 - There must be at least one of the following:
 - Purulent discharge from a superficial infection.
 - Organisms isolated from aseptically obtained wound culture.

Recommendation to minimize surgical site infection

The patient

1. Correct any PDFs as control of diabetes, stopping of smoking and correction of nutritional deficiency.
2. Operations are avoided in patients with active infections if possible.
3. Shaving or clipping the hairs just before skin incision.
4. Skin preparation with appropriate antiseptics.

The surgeon

1. Surgeon should have short nails & should scrub properly.
2. Meticulous surgical technique by proper hemostasis, gentle handling of tissue & avoiding tight sutures or leaving dead space.
3. Delayed primary closure of heavily contaminated wounds.

Prophylactic antibiotics➤ **The choice of prophylactic antibiotics:**

- Select the antibiotics active against the organism likely to be encountered in the hospital.
- Select the antibiotics which give high concentrations in the required tissues used for a suitable time.
- Select the antibiotics which are unlikely to induce renal or hepatic damage if such damage already occurs, correct dosing regimens should be used.
- The evidence that newer expensive broad spectrum antibiotics are more effective for prophylaxis than the cheaper narrower spectrum antibiotics is weak.

➤ **Clean operations:**

- No need for prophylactic antibiotics except in:
 - When an implant, e.g. prolene mesh or a vascular graft is used.
 - In patients with valvular heart disease to prevent infective endocarditis.
 - In emergency surgery in a patient with pre-existing active infection.
 - When infection would be very severe or have life-threatening consequences as in aortic surgery or organ transplantation.

✚ Recommendation:

- One dose of 1st generation cephalosporin, or (ampicillin + sulbactam).

➤ **Clean contaminated operations:**

- One dose of 2nd generation cephalosporine or (ampicillin + sulbactam) + aminoglycoside.

➤ **Contaminated operations:**

- As contaminated, but add metronidazole.
- In all if the operation exceeds 2 hrs, another dose is prescribed.

Treatment

- Liberal drainage. The wound should be opened by removal of skin stitches.
- Antibiotics are used in invasive infections guided by culture & sensitivity tests.
- Possible sources of hospital acquired infection should be traced and corrected.

Decisive period:

- After break in the tissue the acute inflammatory humoral and cellular defenses take about 4 hours to be mobilized. This is called the decisive period and it is the time when invading bacteria become established in tissue so prophylactic antibiotic should be given to cover this period.

⇒ **Systemic inflammatory response syndrome (SIRS):**

- Is the clinical combination of at least two of the following findings:
 - Hyperthermia >38 °C.
 - Hypothermia <36 °C.
 - Tachycardia >90 BPM.
 - Tachypnea >20 /m.
 - WBC >12x10⁹ /L or <4x10⁹ /L.

⇒ **Sepsis:**

Is SIRS with a documented infection anywhere in the body.

⇒ **Severe sepsis (sepsis syndrome):**

- When sepsis is present with evidence of one or more organ failure (Respiratory, Cardiovascular, Renal or C.N.S.).

More details

Asepsis Wound Score

Criteria	Points
Additional treatment	0
Antibiotic for wound infection	10
Drainage of pus under local anesthesia	5
Debridement of wound under general anesthesia	10
Serous discharge ^a	Daily 0-5
Erythema ^a	Daily 0-5
Purulent exudate ^a	Daily 0-10
Separation of deep tissue ^a	Daily 0-10
Isolation of bacteria from wound	10
Stay as in-patient over 14 days as a result of wound infection	5

a- Scored for 5 of the 1st 7 days only the remainder being scored if present in the 1st 2 months.

Blood infection: bacteremia, septicemia and pyemia

↪ Bacteremia

- It is transient condition where organisms circulate in the blood stream without producing any clinical manifestation.
- May occur after surgery on infected sites as dental extraction or tonsillectomy.
- It could be dangerous in patient with cardiac or orthopedic prosthesis or patient with rheumatic heart diseases. Such cases should receive antibiotic prophylaxis before any minor surgical procedure or tooth extraction.

↪ Septicemia

- It implies that infection has escaped from control of the immune system of the patient.
- It is more liable to occur with general debilitated condition and in immune compromise patient.
- It occurs most often due to gram-negative organisms that produce endotoxins, which are released in the circulation when the organism dies.
- Blood culture should be taken as it provides the best evidence of sepsis in such cases.

- Treatment of sepsis:

- Fluid and plasma for circulatory support.
- Operative interference for the drainage of the infection site.
- Antibiotics: started at once empirically and can be modified later on according to the result of blood culture.
- Cephalosporins in combination with aminoglycosides may cover a wide range of bacteria.

↪ Pyemia

- It is due to septic emboli released from a septic thrombus in an inflammatory lesion as in cases of:
 - Heart vegetation on a diseased valve.
 - Lateral sinus in middle ear infection.
 - Cavernous sinus in face infection (dangerous area).
 - Osteomyelitis.
 - Portal vein in appendicitis.
 - Pelvis vein in puerperal sepsis.

Parasitic Infections

Bilharziasis

- Schistosomiasis is an enteric parasitic disease.
- It is common among the rural populations in Egypt.

Causative organism

- Trematode worm.
- Schistosoma hematobium → genitourinary.
- Schistosoma mansoni → intestinal bilharziasis.

Life cycle

- See GIT book (Liver chapter).

Clinical picture

- See GIT book (Liver chapter).

Pathology

- See GIT book (Liver chapter).

Complications

- Defined on the affected system.

Investigations

1. Demonstration of ova in urine & stool.
2. CBC → eosinophilia.
3. Cystoscopy & sigmoidoscopy → Biopsy.
4. Radiological examination.
5. FNABC from liver → shows Bilharzial periportal fibrosis.

Treatment

- **Prevention:** health education → waste disposal & water sanitation.
- **Antibilharzial drugs :**
 - Praziquantel 40mg/kg.
 - Oxamniquine 15 – 60 mg/kg for 1 – 3 days.
 - Metrifonate 7.5 mg/kg 2 – 4 wks.

Filariasis

- **See vascular book.**

Amebiasis

- **See GIT book (Liver chapter).**

Hydatid disease (Echinococcosis)

- **See GIT book (Liver chapter).**

Fungal Infections

Candidiasis

Causative organism

- *Candida albicans*.

Predisposing factors

1. Immune suppression (e.g. AIDS & D.M.)
2. Broad spectrum antibiotics.

Clinical presentation

- Oral lesions (Red spots on mucus membranes → become covered with whitish membrane).
- *Candida* of the esophagus → dysphagia.
- *Candida* of the digestive system → diarrhea.

Treatment

1. Oral non-absorbable antifungal e.g. nystatin for oral candidiasis.
2. Systemic antifungal therapy, e.g. amphotericin B, 5-fluorocytosine & fluconazole

Antibiotics in Surgical Infections

Introduction

a. They are drugs that kill microorganisms but not normal cells.

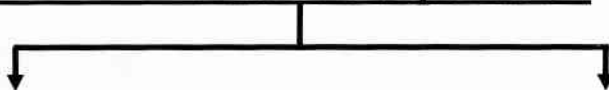
b. **They are two groups:**

Synthetic → As INH, sulpha, quinolones.

Natural →

- They are secreted from organisms to kill other organisms.
- They are either bacteriostatic or bactericidal.

Choice of antibiotic depends on



Patient:

1. Allergy to antibiotic
2. Renal & hepatic function.
3. Resistance of the host
4. Age
5. In female: pregnancy, lactation & other drugs
e.g. oral contraceptive pills.

Causative organisms:

According to C&S

Guidelines before prescribing antibiotics

- Antibiotic should be effective against the most likely pathogen.
- Good diagnosis: samples must be taken & C&S is done.
- The change of antibiotic to another is based on C&S & response of patient.
- Length of antibiotic course is based on pathology, clinical & laboratory assessment.

Common antibiotics in practice

1. β -lactam antibiotics:

- **Mode of action:** inhibit synthesis of bacterial cell wall → lysis.
- **Classes:**
 - a. Penicillins:**
 - **Common agents:** Penicillin G, Penicillin V, Ampicillin, Amoxicillin and Methicillin.
 - **Spectrum:** Gm +ve (mainly) e.g. staph & streptococci & Gm-ve bacteria.
 - Ampicillin lacks activity against some staphylococci but active against enterococci & some Gm -ve bacteria.
 - Methicillin lacks activity against some staphylococci (Methicillin-resistant staph. aureus, MRSA)
 - b. Cephalosporins:**
 - **1st generation Cephalosporins** e.g. cephalothin → active against Gm+ve bacteria but not MRSA.
 - **2nd generation Cephalosporins** e.g. cefamandole → active against Gm-ve but less active against Gm +ve.
 - **3rd generation Cephalosporins:** very effective against Gm-ve but less against Gm+ve infection.
 - Some agents in 2nd and 3rd generations are effective against anaerobes.
 - c. Monobactams:**
 - e.g. Aztreonam → active only against Gm-ve aerobic bacteria.
 - d. Carbapenems:**
 - e.g. Imipenem → active against Gm+ve, most of Gm-ve and anaerobes.

2. Aminoglycosides:

- **Mode of action:** interferes with bacterial protein synthesis.
- **Common agents:** Gentamycin, Tobramycin & Amikacin.
- **Spectrum:** Gm-ve aerobic organisms (mainly) with little activity against Gm+ve and anaerobic organisms.
- **Side effects:** ototoxicity & nephrotoxicity.
- **Precaution:** monitoring of serum creatinine & drug level.

3. Quinolones:

- **Common agents:** Nalidixic Acid, Norfloxacin & Ciprofloxacin (fluroquinolone).
- **Spectrum:** Gm-ve aerobic bacteria (most commonly used in UTI).
- **Side effects:** chondrolytic & nephrotoxic.
- **Precaution:** not used before 18 years old, in pregnancy and lactation.

4. Glycopeptides:

- **Mode of action:** interferes with bacterial cell wall synthesis.
- **Common agents:** Vancomycin.
- **Spectrum:** Gm+ve bacteria even MRSA & anaerobic bacteria (the drug of choice for treatment of antibiotic-induced colitis which is caused by over growth of clostridium difficile).

5. Tetracycline & Chloramphenicol:

- More active against anaerobic organisms.
- Their use in surgical patients has been replaced by more effective drugs.

6. Co-trimoxazole:

- Effective against a variety of Gm-ve aerobic bacteria & some unusual infections in the immunosuppressed patients as those caused by pneumocystis carinii & nocardia asteroides.

7. Metronidazole:

- Excellent anaerobic activity but no effect against aerobic organisms.

8. Erythromycin (Macrolides):

- Active mainly against Gm +ve bacteria & is usually used in patients sensitive to β -lactam agents.

9. Clindamycin:

- Very effective against anaerobes & has some Gm +ve activity.

Complications

1. Hypersensitivity reactions especially with Penicillin and Streptomycin.
2. Vitamin B deficiency due alteration of bowel flora.
3. Superinfection e.g. with proteus and candida.
4. Specific toxicity e.g. bone marrow toxicity with Chloramphenicol.
5. Emerge of resistant strains with mal-use of antibiotics e.g. MRSA.

Antibiotics combination**➔ Examples:**

1. Bacteriostatic + Bactericidal → poor effect.
2. Bacteriostatic + Bacteriostatic → Bacteriostatic effect. Except with trimethoprim, sulphamethoxazole → Bactericidal.
3. Bactericidal + Bactericidal → bactericidal.

➔ The combination may be:

- a. Agonistic (additive or synergistic effect)
- b. Antagonistic.

➔ Indications for combination:**➤ Mixed infections as:**

- Peritonitis due to perforated PU (aerobic, anaerobic).
- After hysterectomy (aerobic, anaerobic).
- We can use Metronidazole+ Ampicillin + Garamycin.

➤ To get synergistic effect of the drugs as:

- Ampicillin + Garamycin.
- Trimethoprim + Sulphamethoxazol.

➤ To overcome bacterial tolerance

- To overcome resistance of staph. → Amoxicillin + Clavulanic acid (Augmentin®) or Ampicillin + Sulbactam (Unasyn®).

➤ To prevent development of resistance

- Treatment of TB → by INH + Ethambutol + Rifampicin.

Decontamination**➔ See operative book.**

Hand Infections Table

	Acute paronychia	Pulp space	Tenosynovitis of middle 3	Ulnar Bursitis	Radial Bursitis	Web Space	Thenar Space	Superficial Mid Palmar	Deep Mid palmar
Definition" inf. of & incidence	Tissue surrounding nail bed commonest	SC compartment of palmer surface of the distal phalynx							
Pathology	Organism: stpah, strept. Route: direct PDF: bad general condition, bad hygiene								
Clinical Picture	Symptoms: General: FAHM Local: pain, swelling, disturbance of function					Signs: general: fever, tachycardia Local: red, hot, edema (dorsum of hand) + enlarged, elastic tender mobile LNs			
Specific Clinical Picture	First: indurated then cystic in shape	First indurated then cystic	Hook sign	Semi-flexion of little finger with max, tenderness kanaval point	Thumb is semi-flexed	Sign of victory		Obliteraion of palm concavity	
Complications	Genera: toxemia, bacteremia et... Local: limitation of movement osteomyelitis Limitation of nutrition osteoarthritis								
Local	Synovial abscess	Hocking stick Parrot peak deformity				Spread to deep mid palmar and other web spaces		Spread + collar stud abscess	
Investigations	1- X-ray: if FB is suspected 2- blood sugar: in recurrent cases may detect DM								
Treatment	1- Before suppuration 2- After suppuration 3- Specific treatment								
Before Suppuration	3 A + Hot fomentation Arm position → Slight → sling → Severe → elevate				Hand position according to expectation - Resolution: ↑ flexion of little finger grasp a ball of cotton ↓ flexion of index - Stiffness : position of function → like holding something				
After suppuration	Once pus is formed do not wait for fluctuation Under general anesthesia or ring anaesthesia 4 Avoid - Avoid: bloody field by limb elevation and calf inflation – Avoid drain .. use tulle-grass - Avoid skin crease and digits midline - Avoid leaving pus or granulation tissue								
Specific: Incision (counter incision if severe)	- Oblique - U-shaped - outer 1/4 Nail extraction	At point of max. tenderness or Anterolateral at distal 2/3 of distal ph. Or Sequestrectomy if parrot peak	(transverse incision) Over cul de Sac along radial border of hypothenar eminence ↓ Ureteric catheter ↓ Irrigation by pinicillin	Along ulnar border of thenar eminence stop proximal 1.5 inch (motor branch of median nerve)	Transverse incision over the web stop 1 cm from free margin	Curved incision along 1 st web space	Transverse incision at line of crease	Transverse incision over the space	

CHAPTER: 7

TRAUMATOLOGY

- 1- Surgical trauma
- 2- Wounds & wound healing
- 3- Blood grouping
- 4- Blood transfusion
- 5- Hemorrhage
- 6- Shock:
 - Hypovolemic shock
 - Septic shock
 - Cardiogenic shock
 - Neurogenic shock'
 - Infusion therapy
 - ARDs

Major Trauma

"Triage" "Poly-traumatized Patients"

Definition

- Trauma is body injury which is accompanied by local as well as systemic effects.

Incidence

- Represent the commonest cause of death among people aged 1-44 years.
- It is the 3rd commonest cause of death among all age groups.
- Number of permanent disabilities caused by trauma is double the number of deaths.

Etiology

1- Closed (Blunt) trauma:

- Direct → e.g. motor car accident (most common), fall from height...etc.
- Indirect: fracture ribs.
- Spontaneous rupture.

2- Open (Penetrating) trauma: stab wounds, iatrogenic...etc.

3- War injury:

a. Gunshot injuries

- Severity of injury depends on velocity of shot.

I. Low velocity

- Entry wound + exit wound (exit wound larger than entry wound).
- Severe tissue contamination from clothes & other foreign materials.
- e.g. hand gun.

II. High velocity

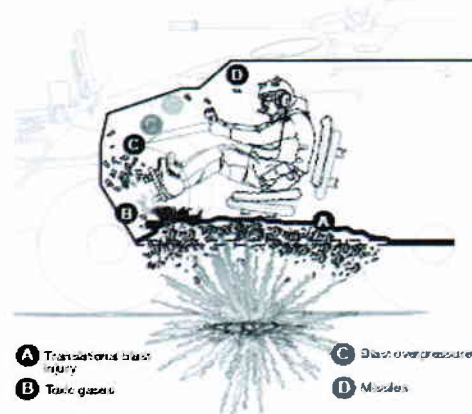
- Explosive pressure + decompression effect.
- Wide spread tissue damage.
- Injury to major limb vessels and nerves.
- Bone fragmentation.

b. Blast injury

Pathophysiology

- Blast injuries traditionally are divided into 3 categories. A patient may be injured by more than one of them.
 - **Primary blast** → injury caused by direct effect of blast overpressure on tissues → always affects air-filled structures such as lungs, ear and GIT.
 - **Secondary blast** → injury is caused by flying objects that strike people.
 - **Tertiary blast** → This type of injury occurs when people fly through the air and strike other objects.

- **Etiology:** closed, open, war inj.
- **Cause of death:** immediate, early, late
- **Triage system:** red, yellow, green
- **TTT:** 1ry & 2ry survey, definitive ttt



Pathology

1- Parenchymatous organs

- Sub-capsular hematoma.
- Superficial or deep tears.
- Avulsion of a pole.
- Complete debulbing.
- Injury of a vascular pedicle.

2- Orthopedics → displacement.

3- Vascular

- With tear:
 - o Types: - Complete → ↓ bleeding. - Incomplete → ↑ bleeding.
 - o Effect: - Artery alone → false aneurysm - Artery & vein → A-V fistula
- Without tear:
 - Spasm.
 - Contusion.

4- Head Injury:

- Rapid acceleration / deceleration and focal forces can all injure the brain.
- Coup injury results from trauma at the site of impact.
- A counter coup injury is remote from the site of impact.

- if a moving car stops suddenly, the person will continue moving forward.
- If he is not wearing the seat belt, the head will strike against the car. However, the seat belt itself may cause injury to clavicle, GIT.
- Seat belt injuries: may include fracture clavicle, trauma to the intestine or mesentery. The skin mark should raise suspicion.

Causes of death:

- **Immediate (minutes):** Hemorrhage, associated injuries to vital structures.
- **Early (hours):** hemorrhage, major fractures.
- **Late (weeks):** sepsis, MOF.

Classification (TRIAGE SYSTEM)

- **Triage** involves sorting patients into three groups by colored labels according to their injury and availability of resources as follow:
 - 1) **Red:** those who will die anyway whether they receive medical attention or not.
 - 2) **Yellow:** those who will survive only if they receive timely medical attention. The first hour after injury is called **golden hour**.
 - 3) **Green:** those who will survive anyway whether they receive medical attention or not.



- If the number of patients exceeds the resources, the yellow code is treated first.
- If the number of patients doesn't exceed the resources, all codes are treated.

Management of Poly-Traumatized Patient

Successful management of poly-traumatized patients require the integration of pre-hospital, in-hospital and rehabilitation which are included in **Advanced Trauma Life Support system (ATLS)** which is a safe and reliable approach for initial assessment and treatment of trauma as follow:

This includes a pre-hospital phase and a hospital phase

ABCDE

Objectives

- To identify & treat any immediately life-threatening condition.

Steps

- A. **A**irway (and cervical spine control)
- B. **B**reathing.
- C. **C**irculation and hemorrhage control
- D. **D**isability. (Brief neurological assessment)
- E. **E**xposure & Environment.

- ➔ **Life threatening problems discovered during the 1ry survey (e.g. tension pneumothorax) are always dealt with before proceeding to the 2ry survey.**

A- Airway (and cervical spine control)

- **Assessment:** - if the patient is able to speak freely, his airway is patent.
- All the patients receive supplemental O₂ by mask upon arrival.
- **Action:**
 1. Clear airway.
 - Vomit blood or foreign material should be removed manually (finger sweep) or with a rigid sucker.
 - This is followed by chin-lift or jaw thrust.
 2. Protect airway:
 - Oropharyngeal or nasopharyngeal airway prevents tongue from falling back & occluding the airway in an unconscious person.
 - Tracheal intubation is indicated with:
 - Apnoea.
 - Risk of aspiration.
 - Impending or actual airway compromise (inhalation injuries, maxillofacial trauma).
 - Closed head injuries (allow hyperventilation to ↓ ICP)
 - Orotracheal intubation allows the use of a large tube.
 - Nasotracheal intubation is safer if the cervical spine appears fractured.
 - Cricothyrotomy:
 - It is not suitable for children

Tracheostomy is rarely needed in the emergency room management

3. Cervical spine control:

- Should be considered unstable if:
 - a. Clinical examination reveals bony abnormalities or cervical tenderness.
 - b. Multisystem trauma, a blunt injury above the clavicle or an altered level of consciousness from trauma or from drug / alcohol intake.
 - c. Maxillofacial trauma.
- Cervical spine immobilization is done using a backboard & a rigid collar.
- If a collar is not available, manual in-line immobilization is necessary.
- Radiological evaluation is done later after stabilization of vital signs, at least 3 views (lateral, AP, odontoid) are done.

B- Breathing▪ **Assessment:**

1. Inspection: for air movement, respiratory rate, cyanosis, tracheal shift, jugular venous distension
2. Palpation: for subcutaneous emphysema & flail segments.
3. Auscultation for upper airway sounds (stridor, wheezing or gurgling).
4. Percussion for hyperresonance or dullness over either lung field.

▪ **Action:**

- Tension pneumothorax → Needle decompression.
- Cardiac tamponade → needle pericardiocentesis.
- Flail chest → intubation & PEEP.
- Massive pneumothorax → thoracotomy & chest tube.
- Open pneumothorax → occlusive dressing & chest tube.

C- Circulation

- **Assessment:** general examination for shock: hemorrhagic (commonest), cardiogenic (tamponade) or neurogenic (spinal cord injury).

▪ **Action:**

1. Control bleeding with direct pressure if possible.
2. Two large-calibre (16 gauge) peripheral IV lines are inserted.
3. Blood samples are sent for typing, cross matching, Hb%, HCT & blood chemistry.
4. Ringer's lactate solution is infused as a start.
5. IV fluids & blood are given.

D- Disability

- **Assessment:** (brief neurological assessment)

▪ Common causes:

- Head injury.
- Hypoxia.
- Shock.
- Alcohol or drug abuse

▪ **AVPU evaluation:** based on patient's best response

- **A:** Alert & interactive.
- **V:** Vocal stimuli elicit a response.
- **P:** Painful stimuli are necessary to elicit a response.
- **U:** Unresponsive.

- Followed by Glasgow coma scale in the secondary survey.

E- Exposure & Environment

- **Clothes:** all clothes of the victim are removed using sharp large scissors.
- **Warmth:** keeping the emergency room warm & using blankets to prevent hypothermia.
- **Insert:**
 - Foley's catheter to monitor urine output (this is contraindicated if there is blood at the urethral meatus).
 - Nasogastric tube (Ryle's) decompresses the stomach & prevents vomiting & aspiration.
- **Radiological assessment:**
 - For blunt trauma → Chest & pelvis X-ray AP view.
 - For penetrating trauma → CXR & X-ray on trauma site.
 - CT scan or X-rays after resuscitation.

■ **History: (AMPLE)**

- Allergies.
- Medications.
- Past medical history.
- Last meal (time).
- Events of injury.

B. Secondary Survey

Objectives:

1. Examination of the patient from head to toe & front to back.
2. Taking a complete medical history.
3. Integration of all clinical, laboratory & radiological information.
4. Formulation of a management plan.

This phase includes:

1- Head to toe examination of undressed & stable patient

- Head examination:
 - Injuries
 - Eye (pupil → size and reaction)
 - Mouth
 - Ear and nose
- Neck & spine: neck collar for fixation:
 - Absent pain or neurological signs does not exclude injury.
 - Move the patient's body as one piece (**log rolling**).
- Chest: pneumothorax, hemothorax, cardiac tamponade.
- Abdomen:
 - Indication of peritoneal lavage:
 - a) Unconscious patient and hypotension of unknown etiology.
 - b) Injury below and above diaphragm and evidence of abdominal injury.
- DRE:
 - Blood in lumen
 - Prostate
 - Pelvic floor
 - Sphincter tone
- Neurological: Glasgow coma scale.
- Limbs: for fractures and neurovascular bundle.

2- History (AMPLE history):

- Allergies
- Medications
- Past medical history
- Last meal
- Event of injury

3- Urgent investigations after basic life support:

- Laboratory:
 - HB%, glucose level, Kidney functions, PO₂, PCO₂, Na⁺, K⁺
- Radiological:
 - Plain X-ray: chest and skeletal or visceral injuries.
 - CT and MRI: chest, abdominal or head traumas.
 - U/S: for abdominal injuries.
 - Duplex for vascular injuries.
- Instrumental:
 - Abdominocentesis or thoracocentesis.
 - GIT or urinary endoscopies.

C. Definitive Treatment

- During secondary survey after stabilization of the patient we can detect the definitive injury by its specific clinical picture and specific investigations and deal with the patient according to the type of injury and priorities.
- The patient require repeated evaluation in some injuries (e.g. spleen, duodenum & subdural hematoma).

Rehabilitation

- Rehabilitation facilitates productive living for disabled people within the community.
- It requires coordination of the patient, medical staff & social services.

Prognosis

- Depends on the severity of the lesion and time passed during arrival to hospital
- Immediate deaths represent 50%, early deaths (1st few hours) represent 30% and only 20% are late deaths due to multiple organ dysfunction syndrome.

Points To Remember

- **At the end of the major survey, detect or exclude the following Five serious problems:**
 1. Airway obstruction
 2. Open or tension pneumothorax
 3. Massive hemothorax
 4. Flail chest
 5. Cardiac tamponade
- **The Five causes for upper airway obstruction:**
 1. Tongue
 2. Blood
 3. Loose teeth or dentures
 4. Vomitus
 5. Soft tissue edema
- **When you face a patient with severe bleeding , remember the five areas of potentially severe bleeding**
 1. Chest
 2. Abdomen
 3. Pelvis
 4. Long Bone Fractures
 5. External bleeding
- **Five main features of respiratory distress**
 1. Tachypnea and/or dyspnea
 2. Use of accessory muscles of respiration
 3. Difficult speaking
 4. Low Oxygen saturation
 5. Agitation or confusion

Metabolic response to trauma

- The response is characterized by an acute catabolic reaction, which precedes the metabolic process of recovery and repair. This metabolic response to trauma was divided into an ebb and flow phase by **Cuthbertson**.
- The **ebb phase** corresponds to the period of severe shock characterized by depression of enzymatic activity and oxygen consumption. Cardiac output is below normal, core temperature may be subnormal, and a lactic acidosis is present.
- The **flow phase** can be divided into:
 - A **catabolic phase** with fat and protein mobilization associated with increased urinary nitrogen excretion and weight loss, and
 - An **anabolic phase** with restoration of fat and protein stores, and weight gain.
- In the flow phase, the body is hypermetabolic, cardiac output and oxygen consumption are increased, and there is increased glucose production. Lactic acid may be normal.

Phase	Duration	Role	Physiological	Hormones
Ebb	<24hrs	maintenance of blood volume; catecholamines	▪ Decrease BMR, Temp, O₂ consumption; vasoconstriction; ▪ Increase Co, incr. HR; acute phase proteins	Catecholamines, Cortisol, Aldosterone
Flow				
<i>Catabolic</i>	3-10 days	maintenance of energy	▪ Increase BMR, Temp., O₂ consumption, negative nitrogen balance	Incr. glucagon, insulin, cortisol, catecholamines
<i>Anabolic</i>	10-60 days	replacement of lost tissue	positive nitrogen balance	Growth hormone, IGF

Renal changes

- ☒ Oliguric renal failure.
- ☒ When acute oliguric renal failure complicates trauma:
 - ♦ Retention of → urea, uric acid, and creatinine
 - ♦ Acid products of cell metabolism such as phosphates and sulphates → cannot be excreted.
 - ♦ Liberation of intracellular K⁺ & Mg⁺⁺ → high levels in extracellular fluid.
 - ♦ Metabolic acidosis → manifested by a decreased CO₂ combining power.

ENDOCRINE RESPONSES TO TRAUMA

- ☒ The adrenal cortex → aldosterone secretion → ↑ sodium and water reabsorption by the renal tubules.
- ☒ ↑ Catecholamines secretion → VC, tachypnea & tachycardia.
- ☒ ⊕ hypothalamus → post. pituitary → ADH secretion → H₂O retention
→ Ant. pituitary → GH, Prolactin & ACTH secretion
- ☒ ↑ IL1 (by macrophages) → ↑ body temperature (fever), WBCs & IgG

Wounds

Definition

- **Wounds:** Disruption of continuity of any body structure by physical injury.

Classification of wounds

A. According to etiology:

1. Closed (blunt): e.g. motor car accident, falling from height.
2. Open (penetrating):
 - a) Accidental e.g. gunshot, cut wounds, stabbing, bites
 - b) Surgical trauma

B. According to extent of tissue injury:

	Tidy wounds	Untidy wound
Mechanism of injury	Incision	Crushing or avulsion
Cleanliness	Clean	Contaminated
Tissues	Healthy	Devitalized
Tissue loss	No or little	Much tissue loss
Healing	1 ^{ry} intention	2 ^{ry} intention

C. According to onset:

- i- Acute wounds
- ii- Chronic wounds

1. Acute Wounds

I- Open Wounds:

- Surgical: Caused by sharp instruments. They are always tidy and cleanly cut.
- Non-surgical.

II- Closed Wounds: with an intact epithelial cover

I- Open wound

1. Abrasions

- **Etiology:** friction with a rough blunt object.
- **Fate:**
 1. Heals with epithelialization → produces no scar.
 2. May cause hyper-pigmentation of the affected area.

2. Puncture Wounds

- **Etiology:** pressure by sharp object (pointed instrument).
- **Character:** The wound depth is greater than its length on the skin surface.
- **Complications:**
 - Possible nerve, vessel or deep viscus injury
 - Needle prick injury carries the risk of serious disease transmission (e.g. HIV and Hepatitis viruses)
 - Foreign bodies deep in the tissues → infection.

- **Class.:** acc. to(etiology, extent, onset)
- **C/P:** pain, swelling, comp.
- **Comp.:** -general: shock, inf., crush s.
-local: inf., inj., comp. of healing
- **Invest.:** ATLS invest.
- **TTT:** 1ry, 2ry, definitive

3. Bites

- **Etiology:**
 - Human bites
 - Animal bites

4. Cut (incised) wounds

- **Etiology:** incising by a sharp cutting object.
- **Character:** The cut edges are cleanly cut and tidy.

5. Lacerations

- **Etiology:** Severe violence with heavy objects.
- **Character:** The wounds are untidy with irregular devitalized edges.

6. Degloving

- **Etiology:**
 - a- Open degloving: e.g. ring avulsion injury with loss of finger skin
 - b- Closed degloving: e.g. rollover injury caused by passage of motor vehicle over a limb.
- **Character:** skin and subcutaneous fat are stripped by avulsion from its underlying fascia, leaving underlying structures exposed.

7. Gunshot wounds

- May be high velocity missile injuries (rifles) or low velocity ones (pistols).
- The damage depends on the following factors:
 1. Direct damage by the missile in its track.
 2. Shock waves : sometimes damage occurs in areas faraway from the missile track.
 3. Temporary cavitational effect.
 4. In the missile strikes a bone, the fragments of shattered bone act as 2ry missiles producing more damage.

II- Closed wound

1. Contusion

- **Etiology:** blunt object producing bleeding into the tissues.
- **Character:** skin discoloration (therefore is called Bruises or Ecchymosis).
- **Fate:** takes variable time for the discoloration to clear off (needs no specific treatment).

2. Hematoma

- **Definition:** Collection of blood in a potential space
- **Clinical picture:**
 - a. A tender cystic swelling.
 - b. In deep parts may be invisible (e.g. in thigh and gluteal region)
- **Complications:**
 - a- Infection → abscess.
 - b- Fibrosis → firm mass.
 - c- Calcification → hard mass.
- **Treatment:** Aspiration using a large bore needle or open surgical evacuation.

3. Compartmental Syndrome

- See orthopedics

4. Crush injuries

- See orthopedics

2. Chronic Wounds

1. **Leg Ulcers** See ulcers

2. **Pressure Sores** See plastic surgery

Clinical Picture

Symptoms

- a- **History of trauma:**
 - Mechanism and extent of injury
 - Other injuries
- b- **Pain**
- c- **Swelling**
- d- **Disturbance of function**

Signs

General

- a- Signs of associated injuries, which may be life threatening.
- b- Signs of complications (shock).

Local

- A- **Examine wound:** (Tidy or untidy)
 - Edges (incised, lacerated and crushed)
 - Floor (clean, contaminated)
 - Tissues around the wound whether healthy or devitalized.
- B- **Examine for complications:**
 - Artery: 6 Ps
 - Nerve: sensory and motor manifestations.
 - Musculoskeletal: loss of special movement.
 - Viscera: e.g. fracture pelvis associated with bladder injury.

Complications

General

- A- **Shock** (hypovolemic, neurogenic, septic)
- B- **Infection:**
 - Specific (tetanus, gas gangrene)
 - None-specific (septicemia, pyemia)
- C- **Crush syndrome:** leading to acute renal failure

Local

- a- **Infection:** factors favoring wound infection:
 - 1- Type of wound (untidy): e.g. puncture wounds and bites.
 - 2- Foreign bodies
 - 3- Ischemia
- b- **Injury of:** nerve, vessel, musculoskeletal and viscera
- c- **Complications of wound healing:**
 1. Early: infection, hematoma and wound dehiscence.
 2. Late: abnormal scars (e.g.: hypertrophic scars, keloids or contractures) → disfigurement
 3. Chronicity (chronic ulcer – sinus formation) with possible malignant transformation

Investigations

- **Polytraumatized patient:** urgent investigations included in ATLS (see surgical trauma)
- **X-ray is indicated in:**
 1. Suspicion of joint disruption or fracture
 2. Puncture wounds to rule out retained foreign bodies in depth of wound

Treatment

- **Poly-traumatized patients:**
 - I. Managed according to ATLS (see surgical trauma).
 - 1^{ry} survey: ABCDE.
 - 2^{ry} survey: examination of the patient from head to toes, resuscitation, investigations, monitoring of patient's response to treatment.
 - **Systemic therapy:**
 - Prophylaxis against **tetanus**.
 - Prophylactic **antibiotics** are prescribed. In deep or lacerated wounds, antibiotics against anaerobic infections should be added.
 - If there is bleeding from the wound, the best way is to stop it by direct local compression. Don't apply a tourniquet except as a temporary measure before taking the patient to the theater.
 - **For Bone injuries:** If a **fracture** is suspected arrange for a plain X-ray and splint the fracture.
 - **For arterial injuries:** Feel the **distal pulses**, if not palpable reassess after treatment of shock. Urgent duplex is mandatory if the pulses are not palpable
- **Management of wound:**
 - Treatment of lacerated wound or crush injury.
 - **Irrigation of wound with saline.**
 - **Wound debridement:** removal of all fragments and devitalized tissues:
 - **Skin:** excision of 1-2 mm from edges.
 - **Fascia:** open tense fascia (won't be closed later to avoid compartmentation).
 - **Muscles:** excise dead muscles.
 - **Bone:** decontamination by curettage or pressure irrigation system.
 - **Nerve:** mark cut nerves for delayed repair.
 - **Blood vessels:** ligation, repair or graft (according to state of injured vessel).
 - **Skin:**
 - **Clean (within 8 hours)** → closure without tension.
 - **Infected (after 8 hours)** → leave it opened with sterile dressing then delayed closure or grafting.
 - Clean & early wound (>6 hrs) → 1^{ry} closure.
 - Contaminated or late → delayed 1^{ry} closure.
 - All missile injuries require exploration.

More details about treatment

▪ Definitive treatment of wound

- i- 1st wound care
- ii- Wound dressing
- iii- Surgical wound management

i- 1st wound care

a. Tidy wound:

- Wound cleaning using liberal amount of antiseptic & normal saline.

b. Untidy

- Requires debridement & hemostasis on one or several occasions to transform it from untidy to tidy e.g. crush wound

☒ Timing of suture application:

1- Primary sutures:

- Applied at the time of injury when dealing with a clean surgical wound or recent wound.

2- Delayed primary sutures: after 5 - 7 days.

- Are applied to doubtful wounds or in late presentation after injury.
- The wounds are left open after surgical excision & daily dressing is done.

3- Secondary sutures:

- Are used where an infected wound is left open for dressing & antibiotics waiting to granulate till it gets clean.

ii- Wound dressing

☞ The ideal wound dressing should have the following properties:

- Maintain a moist environment for healing.
- Remove excess exudates.
- Allow gaseous exchange.
- Impermeable to bacteria.
- Atraumatic on removal.
- Non-allergic.

iii- Surgical wound management

- Reconstruction by skin grafts or flaps in large gaped wounds.

☞ Special types of wounds:

1. Puncture:

- Explore for any FB or deep seated injury
- Screening and prophylaxis against needle stick transmitted diseases

2. Hematoma:

- large hematoma: may require release by incision or aspiration
- Old hematoma: may calcify requiring excision if symptomatic

3. Compartment syndrome:

- See orthopedic surgery

4. Bites:

- Prophylaxis against diseases transmitted by animal bites , anaerobic and aerobic prophylaxis

5. Degloving:

- Debridement with excision of all non-viable skin.
- Split thickness skin grafts can be made from degloved non-viable skin to cover raw areas resulted from debridement

6. High pressure injection injuries:

- Wide exposure → removal of all toxic material → careful debridement of all dead tissue.

➡ In contaminated wounds:

1. Don't do nerve repair.
2. Don't do tendon repair.
3. Don't close the deep fascia.
4. Don't close the skin.

Wound Healing

Definition

- Mechanism by which the body attempts to restore integrity of injured part.

Types

- **Types:** 1^{ry}, 2^{ry}, 3^{ry} intention
- **Etiology:** general, local factors
- **Stages:** ①inflammatory ②lag ③proliferative ④remodeling
- **Comp.:** -Wound(failure, infection)
-Scar(stretch, keloid, hypertrophy)
-Contracture

	1 ^{ry} intention	2 ^{ry} intention	3 ^{ry} intention
Character of wound	- Tidy wounds - Opposed edges - No complications	- Untidy wounds - Gapping of edges - Complicated wounds (e.g. infected)	- Wound initially left open to allow drainage, then when healthy closed by 2^{ry} suturing
Scar	Minimal-strong scar	Extensive-weak scar	As primary
Duration	- Seals in 1-2 days - Heals in 1-2 weeks	Much more time is Needed	Closed when the wound is healthy to avoid bacterial contamination

Factors Affecting Wound Healing

A- General Factors

1- Type of patient:

- Age: old patients have poor wound healing d.t. reduced rate of protein turnover.
- Obesity: obese patients have poor wound healing

2- Medical disease: DM, LCF, CRF lead to poor wound healing

3- Malnutrition.

- Protein deficiency leads to diminished synthesis of collagen & ground substance.
- Vitamin A deficiency leads to deficient epithelization.
- Calcium, Zinc, Copper & Manganese also affect wound healing.

4- Medications e.g. steroids, inhibit wound healing.

5- Debilitating diseases as uremia, jaundice diabetes....etc, delays wound healing.

6- Smoking

B- Local Factors

1- Type of wound (tidy Vs untidy) :

- Mechanism (incision Vs crushing).
- Environment (dry Vs moist*, temperature, pH, infection).
- Tissue loss.

2- Vascularity: a good blood supply (e.g. in the face & scalp leads to nice healing while in a poor blood supply (wounds below the knee) causes delayed healing.

3- Irradiation: impairs wound contraction & granulation tissue formation as it causes ischemia due to EAO.

4- Immobilization: helps healing.

- 5- **Tension:** any increased tension in the wound will lead to ischemia & impaired healing. Sutures under tension , hematoma and infection increase tension inside the wound.
 - 6- **Infection:** delays healing. Fibroblasts compete with bacteria for O₂ & nutrition. Moreover, bacteria secrete collagenolytic enzymes, which destroy collagen fibers.
 - 7- **Foreign bodies & necrotic tissue** impair wound healing.
 - 8- **Adhesion of the wound to a bony surface** prevents wound contraction.
- Impaired venous drainage**, as in post-phlebitic limbs, impairs wound healing

➔ Factors which favor wound infection include:

1. Presence of foreign bodies.
2. Dead tissues.
3. Ischemia.
4. Suturing under tension.

- ➔ After 1 week ➔ the wound has only 3% of its final strength.
- ➔ After 3 weeks ➔ the wound has 20% of its final strength.
- ➔ After 3 months ➔ the wound has 80% of its original strength.
- ➔ The tissues never regain their original tensile strength.

* Dry Vs Moist wound healing

Dry Wound Healing	Moist Wound Healing
▪ Hard to epithelialize. ▪ No wound nutrition. ▪ Scabs delay healing due to desiccation of underlying tissues. ▪ Dead tissues are a good media for anaerobic bacteria.	▪ Easy to epithelialize fast. ▪ Allows wound nutrition. ▪ Prevents scab formation. ▪ Allows granulation through cell migration.

Stages of Wound Healing

1. Inflammatory Phase (1st 4 days)

- The initial response of all tissues to injury, including vascular & cellular responses.

a. Vascular response

☞ It is precipitated by:

- Serotonin and histamine release.
- Activation of kallekrin from kinins.
- Platelet activation factor (PAF).
- Complement factor C5a.

☞ It is composed from:

- **Hemostasis:**
 - ✓ Occurs in 5-10 minutes.
 - ✓ Initial vasoconstriction + activation of the clotting cascade.
- **Vasodilatation:**
 - ✓ Occurs during the first 72 hours.
 - ✓ Responsible for the signs of inflammation (red, hot, tender).

b. Cellular response

- ☞ The cells responsible for the cellular response are:

✓ Macrophages:

- **The main cell in wound healing.**
- Stimulate the division of fibroblasts and new blood vessels.
- They are dominant 3 – 4 days after injury.

✓ **Neutrophils:**

- Migrate through the vessel wall (chemotaxis).
- Facilitate the breakdown of debris and bacteria.

✓ **Mast cells:**

- Release histamine and heparin which facilitate the breakdown of debris and removal of collagen.

2. Lag or Substrate Phase (3 – 5 days)

- Formation of capillary endothelium, muco-polysaccharides and collagen fiber.
- There is no increase in tensile strength.

3. Proliferative Phase (5 – 21 days)**a. Epithelial regeneration**

- Loosening of epithelial cells from their dermal attachment.
- Healing by 1ry intention require small amount of new tissue.
- Healing by 2ry intention require large amount of new tissues.

b. Granulation tissue formation

- Healthy granulation tissue:
 - Beefy **red** in color.
 - Formed of new blood vessels in a collagen network.
 - No offensive discharge.
- Unhealthy granulation tissue
 - Less vascular, edematous, hemorrhagic, has sloughs.

c. Connective tissue repair

- **Fibroblasts** produce collagen and ground substance.
- Vitamin C is essential for normal collagen formation..

d. Wound contraction

- Gradual shrinkage is an attempt to decrease the area to be covered by epithelium.

4. Remodeling phase (≥ 1 year)

- Type III collagen → type I.
- Fibers become lined up along the stress lines of the wound.
- Scar tissue becomes softer and its color fades.

Complications of wound healing**1. Wound failure (wound dehiscence)****2. Stretching of the scar**

3. Hypertrophied scar: the scar is raised above the surface but it remains within the confines of the wound. Within months it may regress. This problem is common in the shoulder and the pre-sternal area.

4. Keloid formation: there's over activity of the healing process leading to excessive scar tissue which is raised above the surface and extends beyond the confines of the original wound. It can follow burns, traumatic or surgical wounds. Persons with dark skin are more prone to keloid formation and there's a familial predisposition. Common at ear lobules, shoulder and pre-sternal areas

Treatment of hypertrophic scar and keloids:

- Continuous pressure by silicone gel sheets → causes ischemia of the small blood vessels leading to diminished activity of the fibroblasts
- Intralesional steroids
- Surgical excision: Recurrence rate after simple surgical excision may reach 80%. To minimize recurrence intramarginal excision of the scar is recommended with intraoperative excision of steroids

5. Contracture: proper positioning of the joint during healing can minimize the deformity

6. Surgical site infection

Normal Healing in Specific Tissues

A- Nerve Healing:

- 1- Distal to wound, Wallerian degeneration occurs.
- 2- Proximal to wound, traumatic degeneration occurs till last node of Ranvier.
- 3- Regeneration occurs, from proximal to distal directed by neurotropism at a rate of 1 mm / day.
- 4- Overgrowth and /or poor approximation may lead to neuroma formation.

B- Tendon healing

▪ Same phases as wound healing but differs in that:

1. Two main mechanisms for delivering nutrients and other blood elements to tendon:
 - a- **Intrinsic:** consists of vincular blood flow and synovial effusion.
 - b- **Extrinsic:** depends on the formation of fibrous adhesions between tendon and tendon sheath
2. Early active mobilization: prevents adhesions limiting range of motions, but in 1st 3-6 weeks the tendon lacks tensile strength, so it must be protected by

Suture Materials

➤ **See operative book.**

Wound Dressings

- ➡ It is used to provide the ideal environment for wound healing & prevent bacterial overgrowth.

Common forms of wound dressings

A. Occlusive dressings:

- Conventional gauze & adhesive plaster → prevention of evaporation, undue dryness & protection.
- Local hypoxia of the wound → stimulates capillary proliferation & favors autolysis of sloughs.
- It is non-suitable for infected wounds.

B. Semiocclusive dressings:

- Permeable to water & vapor.
- Impermeable to bacteria.

C. Alginates dressings:

- Formed of seaweeds → create a moist environment.

D. Osmotic agents:

- E.g. honey, urea crystals & Debrisan

Factors the whole healing reactions

1. Vacuum assisted closure (VAC) → afford -ve pressure → enhances formation of granulation tissue & wound contraction.
2. Increase local hydration of the wound → afford moist environment (moist healing)

Blood Grouping

ABO blood grouping system

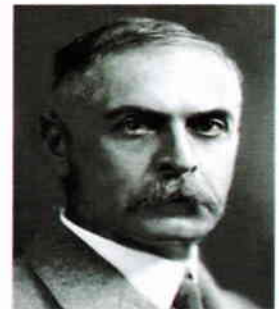
- According to the ABO blood typing system there are four different kinds of blood types: A, B, AB or O.

Blood Group	Antigens (agglutinogens)	Antibodies	Can give blood to	Can receive blood from
AB	A and B	None	AB	AB, A, B, O
A	A	B	A and AB	A and O
B	B	A	B and AB	B and O
O	None	A and B	AB, A, B, O	O

Karl Landsteiner (1868 – 1943)

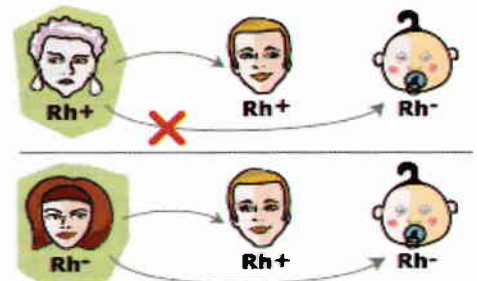
Rockefeller Institute for Medical Research, Austria, New York, NY, USA.

The Nobel Prize in Physiology & Medicine 1930 for his discovery of human blood groups.



Rh factor blood grouping system

- Many people also have Rh factor on the RBCs surface.
- This is also an antigen and those who have it are called Rh+.
- Those who haven't are called Rh-.
- A person with Rh- blood does not have Rh antibodies naturally in the blood plasma.
- But a person with **Rh-** blood can develop Rh antibodies in the blood plasma if he or she receives blood from a person with **Rh+** blood without any problems, whose Rh antigens can trigger the production of Rh antibodies.
- A person with **Rh+** blood can receive blood from a person with **Rh-** blood without any problems.



- "..... In the past, a person with blood type O negative blood was considered to be a universal donor. It meant his or her blood could be given to anyone, regardless of blood type, without causing a transfusion reaction. This is no longer a relevant concept because of a better understanding of the complex issues of immune reactions related to incompatible donor blood cells."

- It is advised for a husband not to donate blood to his wife during her childbearing period. If, after the blood transfusion, the woman develops an antibody to an antigen on the father's red blood cells, and the subsequently born fetus inherits the father's red cell antigen, the antibody from the mother may enter the bloodstream of the fetus causing destruction of fetal red blood cells. This may cause serious anemia in the fetus and excessive jaundice in the infant after birth.

Blood Matching

Direct Matching

Drop of **patient** serum + drop of donor RBCs before transfusion → compatibility is confirmed when no agglutination occurs.

Indirect Matching

Used mainly to determine the blood group.

Blood Transfusion

Definition

- Blood transfusion is the process of transferring of blood or blood-based products from one person into circulatory system of another.

- **Indications:** acc. to component (whole blood, blood derivatives)
- **Comp.:** Immunological, Infection
Stored **blood**, massive **blood**
Cannula (thrombophlebitis, air embolism)

Collection & storage of blood

- Previously it was collected on acid citrate (anticoagulant) dextrose solute (ACD).
- A better anticoagulant is citrate phosphate dextrose.
- One blood bag:
 - 70 – 100 ml anticoagulant (citrate).
 - 400 – 450 ml of blood.
- Blood must be stored at 2 – 6°C during storage.
- Frozen blood:** plasma is removed from freshly collected blood, glycerol is added. The frozen blood doesn't have coagulation factors, platelets or white cells

Blood products & Indications

- Whole blood:** (storage life 2 – 5 weeks)
 - Acute hemorrhage external or internal.
 - Operative and post-operative replacement.
 - Severe burn.
 - Intestinal strangulation.

5. Raising the resistance to sepsis.
6. Exchange transfusion in cases of erythroblastosis fetalis.

II. Blood derivatives :

- a. **Packed RBCs:** (storage life 2 – 5 weeks)
 - Severe anemia & elderly (to avoid volume overload and disease transmission).
- b. **Leucocytes**
 - Severe leucopenia and agranulocytosis.
- c. **Platelets:** (storage life 2 – 5 days)
 - 1^{ry} or 2^{ry} thrombocytopenia and platelet dysfunction (if bleeding or undergoing surgery).
- d. **Plasma:** (storage life 1 – 2 years)
 - **Fresh frozen plasma:**
 - Stored at 30° – 40°C.
 - **Contains:** • Igs.
 - Abs (anti A, anti B Abs, so compatibility is very important.
 - Platelets & coagulation factors.
 - Burn, malnutrition and coagulopathic bleeding e.g. hemophilia, LCF or warfarin overdose (high incidence of blood diseases transmission).
 - **Cryoprecipitate:**
 - Stored at – 40°C.
 - Mainly in hemophilia, DIC & Von Willbrand disease (contains factor VIII & fibrinogen).
 - **Clotting factors concentrates:** According to the deficient factor, e.g. VIII, IX, XI.
 - **Fibrinogen:** hypofibrinogenemia & DIC.
 - **Immunoglobulin:** passive vaccination e.g. anti-tetanic serum.
 - **Human albumin:** hypoproteinemia.

Changes that occurs during blood storage

	0 days	7 days	14 days	21 days
% of red cell viability	95%	90%	85%	75%
% of platelet viability	95%	0%	0%	0%
% of coagulation factor V & XIII	95%	30%	30%	30%
Potassium content (mmol/L)	3.5	10	25	30

Routes of Blood Administration

1. **Intravenous transfusion:** method of choice.
2. Sinus transfusion (rare): mainly in infants via sagittal sinus.
3. Intra-arterial transfusion: life saving in emergency via the radial artery.

Complications

➤ Immunological complications:

1. Incompatible white cells → pyrogenic reaction
2. Incompatible red cells → acute hemolytic reaction
3. Incompatible platelets → purpura
4. Reaction to protein in plasma → allergic reaction

1. Pyrogenic reaction (commonest)

- Due presence of recipient antibodies against some components of donors WBCs
- Patient develops chills, fever, N&V & headache
- Stop the transfusion + give aspirin or paracetamol.

2. Hemolytic reaction

- **Etiology:** incompatible blood transfusion → destruction of donor RBCs by the recipient Ab.
- **Clinical picture**
 - Clinically hemolytic reactions present after 50 ml by fever, chills, constricting pain in the chest, dyspnea & pain in the flanks.
 - A major hemolytic reaction will lead to hemoglobinuria, jaundice & ARF
 - Delayed hemolytic reaction may occur 5-10 days after transfusion in patients who have been immunized to a foreign antigen by a previous transfusion or pregnancy, it presents by unexplained pyrexia or jaundice.

➤ If patient is under anesthesia or comatosed

1. Bleeding tendency (oozing of blood, 1st) (**the most important sign**).
2. Progressive unexplained hypotension.
3. Unexplained tachycardia.

- Treatment of hemolytic reaction

1. Immediately stop the transfusion & repeat patient's blood typing & matching.
2. IV fluid (ringer lactate & IV corticosteroids).
3. Alkalinization of urine by NaHCO₃ (40 mEq).
4. Mannitol 20% 100 ml (**forced alkaline diuresis**).
5. Monitor the urine output and vital data.

3. Post transfusion purpura may develop in patients who have been previously sensitized to foreign platelet antigen

4. Allergic reaction:

- Ranging from urticarial patches up to laryngeal edema.
- TTT: - Antihistaminics & cortisone.
- If reaction is severe → stop transfusion.

5. Infection

- a. Viral → CMV (commonest), hepatitis B & C, HIV.
- b. Bacterial → brucellosis, syphilis (rare now).
- c. Parasitic → malaria (only by red cells, not by blood components).

6. Air embolism

7. Thrombophlebitis at the site of cannula.

8. Complication of transfusion of stored blood:

1. Hyperkalemia.
2. Acidosis.
3. Bleeding tendency

9. **Citrate intoxication:** leading to hypocalcemia. If the patient will receive more than 2 units of blood it is important to administer 10 ml of 10% of calcium gluconate for each 2 units of blood

10. **Transfusion related acute lung injury [TRALI] :** due to incompatibility between donor's Ab & recipient granulocytes. It gives clinical picture similar to ARDS.

11. Massive Transfusion

This implies the transfusion of 2500ml of blood at one time or 5000 ml or more over 24 hours.

- a. Hyperkalemia.
- b. Citrate toxicity → hypocalcaemia.
- c. Hyperkalemia + hypocalcaemia → cardiac arrhythmia.
- d. Acidosis.
- e. Circulatory overload (heart failure).
- f. Hypothermia. A special warming unit should be used to warm the blood before transfusion as hypothermia can cause acidosis or cardiac arrest.
- g. Coagulation failure + platelet dilution → bleeding tendency. Occurs when large volume of stored blood is used to replace blood losses as stored blood is poor in platelets and Factors V and VIII. In these conditions it's recommended to transfuse one unit of fresh frozen plasma and platelets for every unit of stored blood
- h. Diminished O₂ carrying capacity of stored RBCs

N. B.

- Repeated transfusions e.g. thalassemia → hemosiderosis (iron overload).
- Delayed hemolytic reaction may occur after 72 hours but within 2 weeks.
 - Include: delayed hemolysis & post-transfusion purpura.

Indications for transfusion of blood & its products

Product	Indication	Precautions	Storage life
Whole blood	Class III & IV hemorrhage	ABO & Rh	21 days
Red cell concentrates	Severe anemia	ABO & Rh	21 days
Fresh frozen plasma	- Bleeding due to non-specified coagulation factor deficiency. - Coumarin overdose	ABO	1 year at – 40°C
Platelet concentrates	1ry or 2ry thrombocytopenia	ABO	24 – 72 hours
Cryoprecipitates of factor VIII & fibrinogen	Bleeding with fibrinogen depletion	ABO	1 year at – 40°C
Factor IX	Hemophilia A		2 years
Albumin 5% or 20%	- Acute volume expansion. - Hypoalbuminemia.		4 years

Alternatives to homologous blood transfusion

1. Autologous blood transfusion:
 - A patient who is going to have a major elective operation, can donate some units of his own blood over several days.
 - The blood is kept in the refrigerator to be given back to him during surgery.
2. Preservation of the blood lost during surgery & its reinfusion to the patient.
This needs a special apparatus.

How to Avoid Complications of Blood Transfusion

➡ Precautions during collection of blood:

1. Good **selection** of the donor (examination and tests to avoid AIDS & hepatitis).
2. **Sterilized** apparatus and bottle (to avoid infections and febrile reactions).

➡ Precautions during storage and preparation:

1. **Anti-coagulation:** by addition of specific formulas:
 - Previously, **ACD** (acid – citrate – dextrose) → storage for 1 week.
 - Replaced by **CPD** (citrate – phosphate – dextrose) → storage for 3 weeks.
 - Recently, **SAG-M** (saline – adenine – glucose – mannitol) → storage for 5 weeks.
2. **Storage at special refrigerator:**
 - 4°C for blood and packed RBCs.
 - - 40°C for plasma and its fractions.

➡ Precautions before transfusion:

1. Assurance of the **name** of the patient and **date** of storage.
2. **Fresh blood transfusion** (< 6 hours) is recommended in old age, liver disease, renal failure and urgent massive transfusion where the RBCs have temporary reduction to release oxygen to tissue in 24-72 hours.
3. Determination of the **blood group** (direct or indirect method) and Rh antigen.
4. **Cross matching** between the donor's and recipient's blood.

➡ Precautions during transfusion:

1. Don't leave blood outside the refrigerator for more than half an hour.
2. Don't heat the blood except in massive transfusion (using special warming devices).
3. **Use filter apparatus to prevent:**
 - Microscopic clots and debris.
 - Platelet aggregation.
 - Abnormal white cell membrane in stored blood.
4. **Selection of the site** (forearm or dorsum of the hand)
5. **Rate of flow** → 30 drops / minute allows 500 ml / 8 hours (if there is no acute loss).
6. **Give the first 100 ml slowly:** detect immediate reactions (febrile or incompatibility).
7. **Give calcium ampoule** every 100 cc blood

➡ Precautions to avoid over-transfusion: continuous monitoring of CVP.

➡ Precautions during massive transfusion to avoid bleeding tendency: monitoring of clotting should be done by thrombo-elastography in the theater or in the laboratory.

Pathogenesis of renal failure in blood transfusion

- Mismatched transfusion.
- Donor blood itself contains sufficient antibodies to hemolyse recipient cells.
- Intravascular hemolysis leads to an increase in the circulating hemoglobin.
- The hemoglobin is bound by plasma protein and haptoglobin until the amount exceeds 100 mg/100 ml → circulates in the blood → reaches the kidney.
- It causes renal tubular necrosis with subsequent acute renal failure.

Hemorrhage

Definition

- Loss of circulating blood from damaged blood vessels.

Etiology

Traumatic

- Accidental.
- Surgical.

Pathological (spontaneous)

- Blood vessel disease:
e. g. ulceration, infection, malignant lesions or rupture aneurysm.
- Blood disease (hemorrhagic diathesis):
 - Platelet defect.
 - Coagulopathy (e.g. hemophilia, LCF).

Classification of Hemorrhage

1- According to the Source of Bleeding

Venous

- Dark red. (De-oxygenated)
- Continuous & profuse flow.
- Amount depends on the size of the affected vein
- e. g. esophageal varices.

Arterial

- Bright red. (Oxygenated)
- Spurting as a jet.
- Oscillates with pulse.

Capillary

- Bright red. (Oxygenated)
- Oozing, e.g. hemophilia.

2- According to the Time of Bleeding

Primary Hemorrhage

- At the same time of trauma or surgery

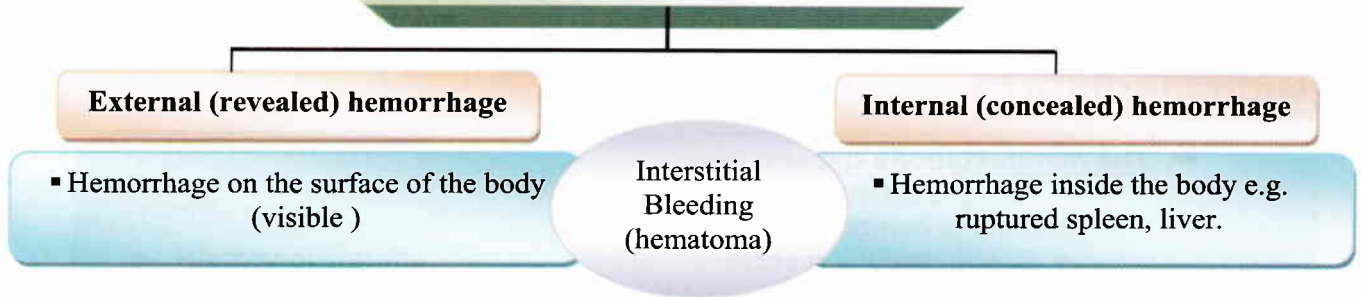
Reactionary Hemorrhage

- Within 24 hours after operation
- Precipitating factors
 - Restoring bl. pressure after surgery
 - Coughing or vomiting
 - Dislodged clot or slipped ligature

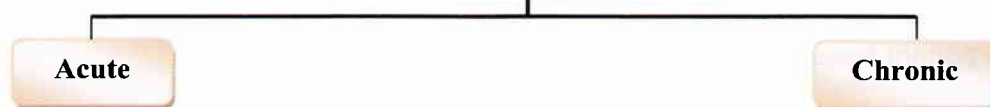
Secondary Hemorrhage

- Within 7-14 days after operation.
- The main cause is infection.

3- According to the Location of Bleeding



4- According to Onset



Physiological response to hemorrhage

1. Stopping the bleeding

- Vasoconstriction and retraction of the intima
- Platelet plug.
- Blood clotting.

These mechanisms occur in sequence. They are more effective when the vessel is completely transected than when there is a side tear, and in traumatic than in pathological cases.

2. Maintaining the effective circulatory volume → To maintain perfusion of brain and heart at expense of muscles, skin and viscera.

A. Neural factors: due to sympathetic stimulation

- Constriction of veins → shifts blood to the arterial circulation.
- Constriction of arterioles → increases peripheral resistance. It involves the arterioles of the skin, skeletal muscle and splanchnic area. Perfusion of heart and brain is maintained because their metabolic needs override the alpha adrenergic vasoconstrictor discharge
- Increased rate and strength of cardiac contraction

B. Endocrine factors:

- Release of catecholamine → increases heart rate and contractility, and cause constriction of the arterioles of the skin, kidney and viscera
- Release of stress hormones e. g. cortisol, glucagon...etc.
- Release of ADH: blood loss greater than 10% stimulates ADH release, it increases the permeability of renal tubules allowing water reabsorption. With severe haemorrhage high level of ADH causes VC
- Activation of renin-angiotensin-aldosterone system → angiotensin II is a powerful vasoconstrictor and stimulates sodium and water retention. Angiotensin mediated VC takes 20 minutes to occur, whereas baroreceptor VC occurs within seconds
- Inhibition of insulin release.

C. Trans-capillary refilling:

- The fall in blood volume → reduces intra-capillary hydrostatic pressure → fluid shifts from interstitial compartment to intravascular compartment → restore blood volume.

Clinical Picture➔ **The manifestations depend upon:**

- Amount of hemorrhage
- Rate of hemorrhage
- Cardiovascular reserve

⇒ Four classes of hemorrhage are recognized based on clinical changes in hemodynamic parameters and indices of tissue perfusion (see table).

➤ **Symptoms:**

1. Weakness and fainting.
2. The patient feels cold and thirsty.

➤ **Signs:**

1. Altered mental status (may vary from anxious to drowsy but the patient usually remains alert).
2. Hypotension, tachycardia and decreased pulse pressure → thready pulse.
3. Hypothermia.
4. Tachypnea and air hunger.
5. Skin becomes pale, cold (VC) and clammy with slow capillary refill (> 2 sec.) and collapsed veins.
6. Oliguria and later on renal failure.

Clinical parameters in different classes of hemorrhage

	Class I	Class II	Class III	Class IV
Blood loss (in 70 kg person)	<i>Up to 15% (750 ml)</i>	<i>15 – 30% (750-1500 ml)</i>	<i>30 – 40% (1500-2000 ml)</i>	<i>> 40% (> 2000 ml)</i>
Mental status	<i>Normal to anxious</i>	<i>Anxious to restless</i>	<i>Aggressive to drowsy</i>	<i>Drowsy to unconscious</i>
Pulse	<i>< 100</i>	<i>100 – 120</i>	<i>100 – 140</i>	<i>> 140</i>
Systolic BP	<i>Normal</i>	<i>Normal</i>	<i>Low</i>	<i>Low</i>
Diastolic BP	<i>Normal</i>	<i>Raised</i>	<i>Low</i>	<i>Low</i>
Pulse pr.	<i>Normal</i>	<i>Low</i>	<i>Low</i>	<i>Low</i>
Resp. rate	<i>14 – 20</i>	<i>20 – 30</i>	<i>30 – 35</i>	<i>> 35</i>
Urine (ml/h)	<i>> 30</i>	<i>20 – 30</i>	<i>10 – 20</i>	<i>0 – 10</i>
Capillary refill	<i>Normal</i>	<i>>2 sec</i>	<i>>2 sec</i>	<i>undetectable</i>

- Loss of 500 ml blood in patient with CAD → hypotension.
- Loss of 1500 ml blood in healthy individual as soldiers → may not lower systolic blood pressure initially

Kasr El-Einy department book

Investigations

(It's an emergency condition, investigations are part of resuscitation)

1. Exclude bleeding tendency: BT, PT, APTT and CBC.
2. HB%, Hct, ABO, Rh → blood transfusion.
3. ABG, PH, electrolytes.
4. KFTs & LFTs.
5. Measurement of CVP (normally 15cm.H₂O).
6. Search for:
 - Associated fracture → X-ray.
 - Rupture viscera → Abdominal U/S.
 - Head injuries → CT scan.
 - Blood vessels injuries → Doppler & Duplex.
 - Hemothorax → Thoracocentesis.

Treatment

1. Stop hemorrhage: (**position - pressure - packing**) e.g.

- **Elevation of the limb** above the heart level stops venous bleeding and decreases arterial bleeding.
- **Pneumatic anti-shock garment (PASG)** → tamponade lower limb, pelvic and abdominal hemorrhage.
- **Balloon tamponade** for hemorrhage from esophageal varices.

2. Restore blood volume: through a wide bore cannula or a cut down on the long saphenous vein, if necessary. The volume depends on the class of hemorrhage:

➡ **Class I:** no replacement.

➡ **Class II:**

- **Estimation of deficit:** 15-30% (750-1500 ml/70 kg).
- **Type of fluid:** Ringer lactate, to restore the intravascular and interstitial fluid.
- **Volume of replacement** = 3 times the estimated deficit (≈3L).
- **Administration:** 2 liters are given as a bolus and the response is monitored.
 - ➔ If there is definite improvement → the remaining liter is given slowly.
 - ➔ If there is moderate improvement, the possibilities are: Inadequate fluid replacement, persistent hemorrhage (start blood transfusion) or myocardial insufficiency (exclude cardiac tamponade and tension pneumothorax).

➡ **Class III and IV:** as for class II + blood transfusion.

- **Estimation of deficit:** > 30% (> 1500 ml)
- **Volume of replacement** = estimated deficit (≈ 1500-2000 ml).
- **Administration:** continue until the hematocrit reaches 30%, the urine output is 50 ml/hour, and the CVP rises to the upper half of the normal range.
 - ➔ Failure to improve with a rising CVP → tension pneumothorax, cardiac tamponade, or cardiac failure (if these are excluded and the patient does not improve, major thoracic, abdominal, or pelvic injury is usually present).

3. Optimize oxygen delivery: 40% oxygen is given for class II hemorrhage and 100% for classes III and IV.

4. **General care of the patient:** absolute bed rest and analgesia (morphia, 3-5 mg IV), repeated if necessary. Morphine is contraindicated in head injury and in cases of respiratory and liver insufficiency.
5. **Monitoring:**
 - Urine output, core temperature, hematocrit and cardiac monitoring (ECG for early detection of shock-induced arrhythmias is important).
 - In class III or IV hemorrhage → as above + central venous pressure (CVP), blood gases and pH.

➤ **Management of bleeding tendency:**

- Platelet transfusion if $< 50,000/\text{ml}$.
- Vitamin K, fresh frozen plasma or cryoprecipitate → coagulopathy.

More details

• **Estimating blood loss:**

1. **Clinical data:** four classes of hemorrhage are recognized.
2. **Type of injury:** a hematoma around a closed fracture of the tibia may contain 500-1500 ml of blood while that around a fractured shaft of femur, 500-2000 ml and that in a fractured pelvis, 2000-3000 ml.
3. **Blood loss at operation** = the amount in the suction reservoir + the amount mopped up by the swabs. The latter is calculated as the difference in swab weight after and before operation $\times$ a correction factor of 1.5-2 (depending on the magnitude of the operation).
4. **During operation:** a blood clot with the size of a closed hand fist = 500 cc approximately.

Haemostasis & Bleeding disorders

Definition

- It is the arrest of bleeding from an injured blood vessel.
- It requires the combined activity:
 1. Vessel walls.
 2. Platelets.
 3. Clotting factors.
- In addition to normal haemostasis, the surgeon can control bleeding by different methods that are called surgical haemostasis.

Natural haemostasis

Primary haemostasis

- Arrest of blood within 1st few minutes → is achieved by:
 1. Vasoconstriction.
 2. Platelet plug formation (platelet adhesion & aggregation).

Secondary haemostasis

- After the 1st few minutes = coagulation → is achieved by two mechanisms

Intrinsic pathway

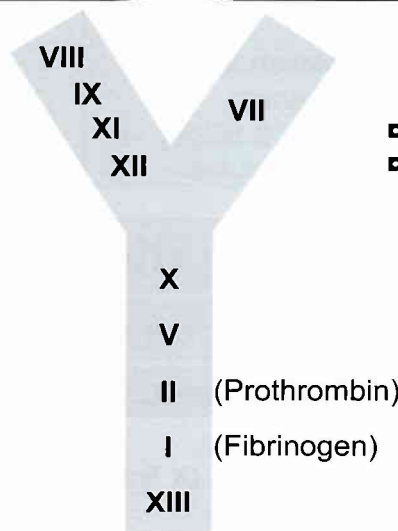
- Within few minutes
- Controlled by PTT

(Heparin mainly affects intrinsic pathway).

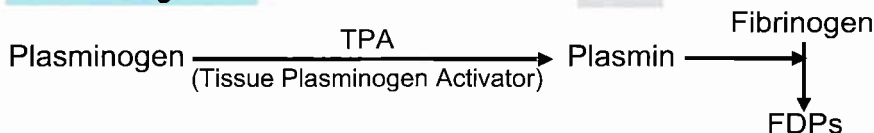
Extrinsic pathway

- Rapid within few seconds
- Controlled by PT

(Oral anticoagulants mainly affects extrinsic pathway)



Fibrinolysis



Bleeding	Platelet count
• Spontaneous bleeding	10,000 – 20,000 /L
• Bleeding with minor trauma	20,000 – 50,000 /L
• Bleeding with major trauma	50,000 – 100,000 /L
• No haematic abnormality	> 100,000 /L

➔ Inhibitors of coagulation:

1. Anti-thrombin III (activated by heparin).
2. Protein C & S.
3. Factor V (Leiden)

Coagulation screening tests

	Times	Disorders
1. Platelet count	Normal = $150 - 400 \times 10^3$	$> 100.000 =$ Thrombocytopenia
2. Bleeding time	< 8 minutes	• Thrombocytopenia. • Abnormal platelet function. • Deficiency of Von Willbrand factor • Vascular abnormality
3. PT	12 – 15 seconds	Deficiency of factor II, V, VII or X (extrinsic & common pathways) → assess oral anticoagulants
4. PTT	30 – 40 seconds	• Deficiency of factor II, V, X, VIII, IX, XI, XII (intrinsic & common pathways) → control of heparin.
5. INR	Ration between $\frac{\text{Patient PT}}{\text{Control PT}}$ Normal = 1	• Patient receiving oral anticoagulants (2 – 3) • Patient with prosthesis (2.5 – 3.5).
6. Others	• Fibrinogen. • FDPs. • Coagulation factors. • Bone marrow aspiration & biopsy → Megakaryocytes.	•

Bleeding disorders

A-Congenital disorders

I- HEMOPHILIA A & B

Definition

- It is an inherited coagulation defect (X linked recessive) due to deficiency of factors
XIII → A
IX → B

Clinical picture

1. Bleeding is post-traumatic since birth (cephalhematoma).
2. Deep hematoma or recurrent hemoarthrosis.

Investigations

- Factors level is 5 – 20 % of its normal value.

Treatment

1. Infusion of deficient factors concentrated within 1 hr before surgery.
2. Fresh frozen plasma & cryoprecipitate.
3. Avoid aspirin, NSAIDs & IM injections (because large hematoma can develop).

II- VON WILLEBRAND DISEASE**Definition**

- It is an autosomal dominant disease with variable expression due to deficiency of VW factor which enhances:
 - Plasma adhesion (1ry).
 - Carrier for factor VIII (2ry).

Clinical picture

- Purpuric eruptions.
- Ecchymosis.
- Bleeding from body orifices.

Investigations

1. Prolonged both PT & PTT.
2. Deficiency of factor VIII & VW factor.

Treatment

- Infusion of deficient VW factor.

B-Acquired disorders

- They are more prevalent.

I- HEPATIC DISEASES

- Acute or chronic liver disease associated with haemostatic abnormalities

Etiology

1. Deficiency of coagulation factors, fibrinogen (PT & PTT are prolonged with ↑ INR).
2. Thrombocytopenia if associated splenomegaly or hypersplenism.
3. ↓ synthesis of inhibitors of fibrinolysis e.g. α_2 antiplatelets.

Treatment

1. Vit. K.
2. FFP (2 – 3 units).
3. Desmopressin ↑ factors VIII, VW & shorten bleeding time.
4. Antifibrinolytics or dexomercaptpurine.

II- VITAMIN K DEFICIENCY

- Vit. K is a cofactor for activation of factor II, VII, IX & XI

Etiology

1. Inadequate diet or TPN.
2. Prolonged use of broad spectrum antibiotics (↓ colonic bacteria).
3. Cholestatic jaundice.
4. Malabsorption.
5. Oral anticoagulants.

Clinical picture

- Easy bruising & traumatic bleeding.

Investigations

- ↑ PT

Treatment

1. Vit. K by slow infusion (5-10 mg/d /or slow IM injection 2 or 3 days [10-20 mg/day]).
2. In emergency: factor concentrates II, VII, IX, X , FFP.

III-DIC

Definition

- It is simultaneous widespread occurrence of microvascular bleeding & activation of coagulation (leading to consumption of coagulation factors) which in turn worsens the bleeding (↑ FDPs worsens the coagulopathy).

Etiology

1. Septicemia.
2. Severe shock, trauma & burns.
3. ABO incompatibility transfusion.
4. Malignancies e.g. metastatic carcinoma of the liver, pancreas & stomach.
5. Obstetric accidents (eclampsia, abruptio placenta, AF embolism).

Clinical picture

Diagnosis is suspected by

1. Diffuse bleeding from wounds, incisions, orifices.
2. Widespread bruises, purpura, mucosal bleeding.
3. Ischemic manifestations in skin & digits.

Investigations

- Low platelet count, ↑ PT, PTT, ↓ fibrinogens, ↑ FDPs.

Treatment

1. Of the cause: e.g. drainage of an abscess, antibiotics for infection.
2. FFP & cryoprecipitate or platelet transfusion.
3. Heparin is not adjusted.

IV- OTHER CAUSES

3. Anticoagulants → bleeding if not properly adjusted.
4. Massive blood transfusion.
5. Platelet disorders:
 - Thrombocytopenia e.g. ITP or 2ry.
 - Platelet dysfunction: aspirin, NSAIDs, anemia.

Preoperative evaluation of haemostasis

History

1. +ve family history of bleeding.
2. History of bleeding tendency.
3. Age of onset of bleeding (congenital or acquired).
4. Liver disease, CRF, massive blood transfusion or drug intake.
5. Criteria of bleeding:
 - 1ry haemostasis: bleeding is superficial, petichae & purpura.
 - 2ry haemostasis: bleeding is deep, hematoma, hemorrhage.
 - Excessive fibrinolysis.

Examination

- If suspecting bleeding tendency.
 1. Cutaneous signs of liver disease.
 2. Skin & m.m. for bleeding, petichae, hematoma.
 3. Cavernous hemangioma → thrombocytopenia.

Investigations

 See before

- Excessive operative & postoperative bleeding is due to:
 1. Inadequate surgical haemostasis (**the commonest cause**).
 2. Hemorrhagic diathesis not detected preoperative.
 3. Acquired intraoperative coagulopathy.

Management

1. Proper surgical hemostasis.
 2. Correction of hypothermia.
 3. FFP, platelet transfusion unless needed.
- **Surgical hemostasis by:**
 1. Ligation: a ligature tied out.
 2. Under running suture.
 3. Electrocautery:
 - a. **Monopolar diathermy:**
 - It is called monopolar because the electric current passes through the patient body between two electrodes. One electrode (passive) is broad & is placed over the L.L. & the other (active) is usually a forceps with a fine tip which the surgeon uses to grasp the bleeding point.
 - As the current passes between the electrodes, it becomes concentrated at fine tipped forceps, this generates heat which causes tissue coagulation.
 - b. **Bipolar diathermy:**
 - Also the current passes between the two blades of one forceps which the surgeon uses to grasp the bleeding point.
 - c. **Clips.**
 - d. **LASER**
 - e. **Repair of injured large vessel.**

Blood disorders

Classification

1. Anemia.
2. ↑ Hb (polycythemia).
3. Leucopenia.
4. Leucocytosis.
5. Lymphadenopathy.
6. Splenomegaly.
7. Bleeding.
8. Thrombocytopenia.
9. Thrombocytosis.
10. Venous thrombosis.
11. Abnormal coagulation.
12. Pancytopenia.
13. Infection.

What are the classifications of anemia?

a. According to the cause

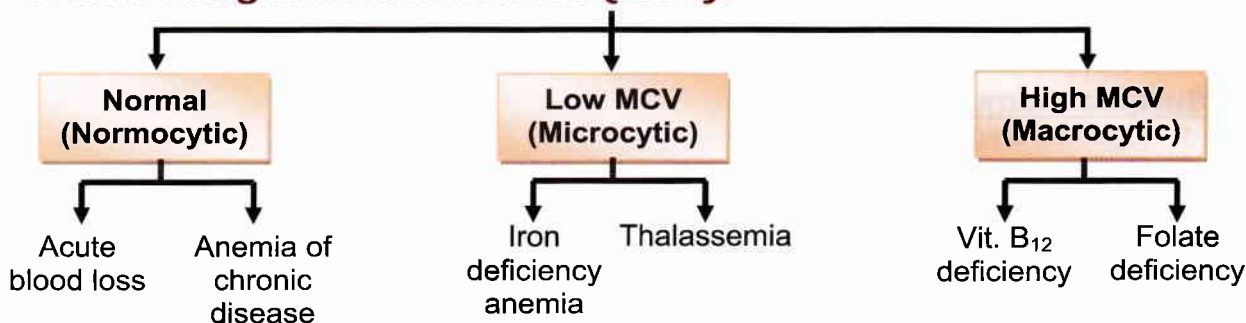
1- Decreased & ineffective B.M. production:

- ▣ Lack of free B₁₂, folate.
- ▣ Renal failure.
- ▣ Hypoplasia.
- ▣ Anemia of chronic disease.
- ▣ Invasion by malignant cells.

2- Peripheral causes due to:

- ▣ Blood loss.
- ▣ Haemolysis.
- ▣ Hypersplenism.

b. According to mean cell value (MCV):



- ▣ The commonest is iron deficiency anemia.
- ▣ The most serious is hemolytic anemia.

Hemolysis

A. Features of anemia:

- ▣ Symptoms: palpitation, head & easy fatigability.
- ▣ Signs: pallor, tachycardia.
- ▣ Investigations: ↓ Hb.

B. Features of hemolysis:

- ▣ By investigations:
 - ↑ Total & direct bilirubin.
 - ↑ CDH
 - ↑ Reticulocytes.

■ **Causes:**

A- INTRINSIC (INTRACORPUSCULAR) → inherited since childhood

1. Membrane defect → hereditary spherocytosis.
2. Hemoglobinopathies → sickle cell anemia.
3. Defect in globin synthesis → Thalassemia.
4. Enzyme deficiency → G6PD & pyruvate kinase deficiency.

B- EXTRINSIC (EXTRACORPUSCULAR):

1. Autoimmune
2. Mechanical → prosthetic valve or microangiopathy.
3. Infection → malaria or clostridia.
4. Chemicals → drugs or snake venom.
5. Hypersplenism.

I- HEREDITARY SPHEROCYTOSIS

Etiology

- AD disease due to defect in cell membrane protein called spectrin → Na-K⁺ ATPase pump & destruction of RBCs in spleen with increase osmotic fragility.

Clinical picture

1. Features of anemia
2. +ve F.H.
3. Features of hemolysis e.g. jaundice, splenomegaly.
4. Pigmented gall stones.

Investigations

1. MCV → normal.
2. Investigations of hemolysis (as above).
3. Osmotic fragility test → +ve.
4. Blood film → spherocytes.

Treatment

1. Splenectomy (good response & improves the condition).
2. Blood transfusion.
3. Folic acid 1mg/day.

II- THALASSEMIA (α & β)

Beta thalassemia

Definition

- Inherited disease due to defective synthesis of β chain of Hb molecule:
- Homozygous (major).
 - Heterozygous (minor).

Clinical picture

1. After 6 m of life due to presence of HbF
2. Features of hemolysis: anemia, jaundice, splenomegaly.
3. Mongoloid features due to marked expansion of B.M.
4. Marked HSM due to extramedullary hematopoiesis.

Investigations

1. Anemia: ↓ Hb, ↓ MCV (microcytic).
2. Hemolytic: ↑ indirect bilirubin.
3. Hb electrophoresis: ↑ HbF & ↑ HbA₂.

Treatment

1. Blood transfusion.
2. Desferal®.
3. Folic acid.
4. Splenectomy & blood transfusion exceeds 250 packed RBCs/Kg ml.
5. B.M. transplantation.

III- SICKLE CELL ANEMIA

Definition

- AR inherited disease due to gene mutation with substitution of glutamine to valine a.a. at position 6 of β -globin chain.

Pathophysiology

- On deoxygenation → polymerization of Hb molecule & sickling of RBCs.

Clinical picture

- Precipitated by hypoxia, dehydration, infection & acidosis → crises & chronic organ disease
1. **C/P of crises:**
 - a- Vaso-occlusive crises (**the commonest**): → bone aches especially in hands & feet, tachycardia, sweating & fever.
 - b- Sick chest syndrome:
 - The most common cause of death in adults.
 - Pulmonary infarctions → severe chest pain → respiratory failure.
 - c- Sequestration crises: thrombosis in venous outflow of organs causes acute painful enlargement e.g. massive splenomegaly → anemia & circulatory collapse.
 - d- Aplastic crises → due to infection by erythro virus 19.
 2. **CVS:** CV accidents & fits.
 3. **Ocular:** background proliferative retinopathy & vitreous hemorrhage.
 4. **Cardiac:** cardiomegaly
 5. **Pulmonary:** sickle chest syndrome.
 6. **Others:**
 - Osteomyelitis
 - Splenic infarctions
 - Cholestiasis.
 - Renal (CRF, hematuria, osteoporosis, papillary necrosis).
 - Pr. (leg ulcers).
 - Dactylitis (infarction of digital blood vessels of fingers & toes).

Investigations

1. Anemia: ↓ Hb, normal MCV (normocytic).
2. Hb electrophoresis: HbS.

Treatment

1. Blood transfusion & folic acid.
2. Crises:
 - a- Analgesics
 - b- NaHCO₃, rehydration by fluids.
 - c- Fresh blood exchange transfusion.
3. Avoid precipitating factors e.g. hypoxia, dehydration & infection.

IV- G6PD DEFICIENCY

- It is an X – linked disease.

Etiology

- Due to deficiency of enzyme involved in hexose mono-phosphate shunt which is the source of NADPH^+ that protects RBCs against oxidative stress as it increases the reduced glutathione that resists oxidation.

Clinical picture

1. Drug induced hemolysis: aspirin, sulfa, quinolones.
2. Chronic hemolytic anemia.
3. Neonatal jaundice.

Investigations

1. Anemia
2. Decreased enzyme level (G6PD).

Treatment

1. Avoid drugs.
2. Blood transfusion & folic acid.
3. Splenectomy.

Disorders of hemostasis**I- Disorder of 1ry hemostasis**

- 1- Thrombocytopenia.
- 2- Platelet function disorders.
- 3- Vessel wall abnormality.
- 4- Von Willbrand disease.

II- Disorder of 2ry hemostasis (Coagulation defects)

- See before

1- Thrombocytopenia**a. B.M. disorders:**

- Hypoplasia e.g. idiopathic, drug induced, cytotoxic.
- Infiltration e.g. leukemia, lymphoma.
- Vit. B_{12} , folate deficiency.

b. ↑ consumption of platelets or destruction:

- DIC.
- ITP.
- Viral infections (EPV & HIV).
- Gram –ve septicemia.
- Hypersplenism.
- Thrombotic thrombocytopenic purpra.
- Liver disease.
- Connective tissue disorders e.g. SLE.

2- Vessel wall abnormality (Non thrombocytopenic purpra)**➤ Types:**

- a- Senile purpra.
- b- Henoch shonlien purpra (HSP).
- c- Factitious.
- d- Vasculitis:
 - Hereditary hemorrhagic telangectasis.
 - Ehlar danlos syndrome.
- e- Drug induced.
- f- Purpra fulminans.

➔ Purpura:

Multiple spontaneous capillary bleeding in skin & m.m. due to defects in:

- Platelets → not raised.
- Capillary wall → raised

3- Platelet function disorders

4- Von Willebrand disease (See before)

ITP

Etiology

- It is an autoimmune disease, where an auto Ab (IgG) attacks platelets causing its destruction

Clinical picture

- Varies according to age.
- May be asymptomatic.

a. In children:

- Acute ITP → bleeding tendency:
 - Skin petichae & purpura without raised edges.
 - Bleeding per orifices & m.m.
 - Internal organ hemorrhage (e.g. cerebral hemorrhage).
 - No splenomegaly.
 - Platelets < 20,000

b. In adults:

- Chronic ITP → minimal or no bleeding tendency
 - Splenomegaly.

Investigations

1. ↓ Platelet count.
2. B.M. examination → hypoplasia of megakaryocytes with defective budding.
3. Abs against platelets.

Treatment

a. In children:

- 1- If asymptomatic (pl. > 40,000) → no need, it is self-limiting condition.
- 2- Platelets 20,000 – 30,000 → prednisolone.
- 3- Platelets < 20,000 → platelet transfusion & IV Ig.

b. In adults:

- 1- Prednisolone (2-3 m).
- 2- If failed → splenectomy (not effective).

Shock

Definition

- **Pathological:**
 - A state of peripheral circulatory failure. Impaired tissue perfusion results in disturbed cellular metabolism.
- **Clinical:**
 - 20% sudden blood loss or shift from peripheral circulation.

Classification

- Adequate blood flow to the tissue depends on balance between:
 - 1- Blood volume.
 - 2- Myocardial contractility.
 - 3- Peripheral resistance.
- A serious disturbance in any of the three components can lead to shock.
- In the form of:
 1. **Hypovolemic "oligemic" shock:** due to diminished blood volume.
 2. **Cardiogenic shock:** due to inefficient myocardial function.
 3. **Distributive shock:**
 - a. **Septic shock:** (see later).
 - b. **Neurogenic shock:** due to peripheral vasodilatation, reduced peripheral resistance and peripheral pooling of blood.
 - c. **Anaphylactic shock:** due to antigen antibody reaction that also leads to peripheral pooling of blood e.g. injection of anti-tetanic serum.
 - d. **Endocrinal shock:** due to acute severe hormonal imbalance. It may present with a combination of any of the above types.
 4. **Obstructive shock:**
 - Cardiac tamponade & pulmonary embolism & tension pneumothorax .

Pathophysiology

A. Cellular:

- Tissue hypoperfusion → ↓ O₂ delivery to the tissues → anaerobic metabolism → ↑ production of lactic acid → metabolic acidosis.
- This continues until cellular glucose is depleted → stoppage of cell metabolism → release of auto-digestive enzymes → Rupture of plasma and lysosomal membranes .

B. Microcirculatory changes:

- Under the effect of catecholamines the precapillary sphincters constrict .Less blood enters the capillaries and the capillary circulation becomes sluggish.
- Untreated hypovolemia thus leads to hypoxia.
- Under normal conditions **only one third** of the capillary bed is open at a time. Under ischemic conditions however the body reacts by **opening more capillaries** .
- **The results are:**
 - Further slowing of the capillary circulation , the so called **slugging** of blood.
 - **Slugging** encourages spontaneous coagulation of blood in these capillaries
 - If extensive , it is called **disseminated intravascular coagulation (DIC)** .
 - This depletes coagulation factors and induces bleeding in the rest of the body (consumption coagulopathy).

- A sluggish circulation, and DIC compound tissue hypoxia and affect the function of capillary endothelium. The result of the latter is leakage of large protein molecules from the vessels into interstitial space dragging with them huge amounts of fluid. This third space loss of fluid further reduces blood volume.

C. Acid-base imbalance:

- **Shock is accompanied by metabolic acidosis**, which is caused by :
 1. Accumulation of lactic acid as a result of anaerobic metabolism.
 2. Renal failure as a result of prolonged ischemia , aggravates acidosis.

D. Systemic:

a) Sympatho-adrenal response:

- Release of catecholamine → causes of redistribution of blood, diversion of blood from non-vital organs (skin, GIT) by V.C. to vital organs by V.P. (coronary Vs) & Ms
→ Increases heart rate and contractility.
- Release of stress hormones e. g. cortisol, glucagon...etc.
- Release of ADH
- Activation of renin-angiotensin-aldosterone system → vasoconstriction – Na⁺ & water retention.

b) Individual organs

▪ Renal:

- Renal hypoperfusion → decrease the GFR → decreased urine output → may lead to pre-renal failure and acute tubular necrosis.

▪ Respiratory:

- Metabolic acidosis → tachypnea → wash out of CO₂ → compensatory respiratory alkalosis.
- Leakage and swelling can develop in the lungs, causing difficulty in breathing (respiratory distress - ARDS).

▪ GIT:

- Mucosal hypoperfusion → mucosal death → stress ulcer.
- Mucosal death → bacterial translocation → septicemia & multi-organ failure.

▪ Cardiovascular:

- Myocardial contractility is impaired by:
 - i. Reduction in coronary arteries perfusion
 - ii. Direct myocardial depressants as cachectin (TNF).

▪ Liver:

Ischemic hepatic dysfunction is a frequent component of MOSF.

➡ Ischemic perfusion syndrome

- It is due to systemic hypoperfusion + tissue hypoxia + local activation of inflammatory mediators.
- Further injury occurs once normal circulation is restored to tissues:
 1. Myocardial depression.
 2. Cellular & humoral elements activated by hypoxia are flushed back into circulation → more endothelial injury, e.g. acute lung injury, acute renal injury, MOF.
- It is only can be minimized by decreasing the period of hypoperfusion.

1- Hypovolemic Shock

"Oligemic" "Hemorrhagic Shock"

Etiology

- **Diminished blood volume:** due to
 - 1- Whole blood loss in hemorrhage "traumatic or pathological".
 - 2- Plasma loss as in burn or peritonitis.
 - 3- Water and electrolytes loss (especially Na^+) as in:
 - Severe vomiting, diarrhea
 - Intestinal obstruction.

- **Etiology:** ↓ (blood, plasma, H_2O)
- **C/P:** ↑ (pulse, RR), ↓ (Bp, temp., urine)
- **Invest.:** as ATLS
- **TTT:** -1st aid
 - definitive ttt: ①Stop hge.
 - ②Resuscitation ③Fluids
 - ④Monitoring ⑤Drugs

Stages

There is no sudden transmission from one stage to the other

1. Compensated Stage

- In this stage, the body employs the physiological mechanisms, in a trial to restore blood volume or at least, preserve the normal function of the vital organs (brain, kidneys, heart and lungs).
- With loss of > 15% of blood volume, the compensatory mechanisms usually fail.

2. Decompensated Stage (40% blood loss)

- If cause of the shock is not treated successfully & after failure of compensatory mechanisms → Progressive poor perfusion & micro circulatory changes.
- Deterioration of functions of brain, kidney, heart and lung.

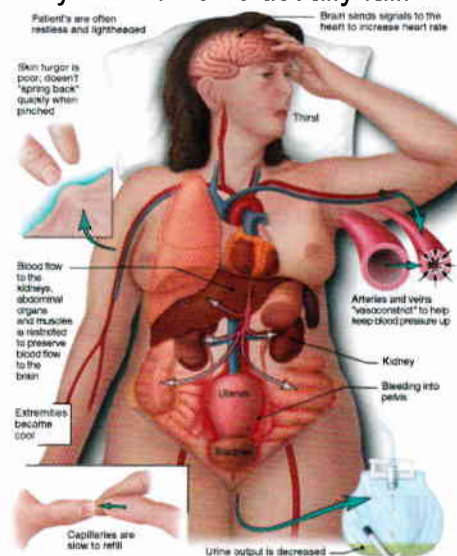
3. Refractory Stage

- The shock can no longer be reversed.
- Failure of vital organs & death occurs imminently.

➔ How much of your 5 liters blood you can lose at once:

- 10% as a donation, thank you.
- 20% you feel little sick.
- 40% you will probably go into hypovolemic shock

Kasr El-Einy book



Clinical Picture

A. Of shock:

▪ Symptoms:

1. Weakness and fainting.
2. The patient feels cold and thirsty.

▪ Signs:

1. Altered mental status (may vary from anxious to drowsy but the patient usually remains alert).
2. Hypotension, tachycardia and decreased pulse pressure → thready pulse.
3. Hypothermia.

4. Tachypnea and air hunger.
5. Skin becomes pale, cold (VC) and clammy with slow capillary refill (> 2 sec.) and collapsed veins.
6. Oliguria and later on renal failure.

B. Of the cause:

- Sites of bleeding.
- Signs of internal hemorrhage.
- Burns.
- Intestinal obstruction

C. Of complications:

- Anuria.
- ARDS.

Investigations

(It's an emergency condition, investigations are part of resuscitation)

▪ Blood sample for:

1. Exclude bleeding tendency: BT, PT, APTT and CBC.
2. HB%, Hct, ABO, Rh → blood transfusion.
3. ABG, PH, electrolytes, KFTs & LFTs.

- Should be done serially as it may be normal at 1st then decreases due to IV fluids & trans-capillary refilling.

▪ To detect the cause:

- ECG → Exclude cardiogenic causes
- Abdominal U/S → Internal hemorrhage.
- Head CT scan → if patient with head injury or unconscious.

Treatment

(Immediate intervention is necessary to guard against fatal outcome).

(Investigations should be done after resuscitation)

➤ Outside hospital or in ER (1st AID)

▪ Airway: should be patent by:

- Pulling the tongue forward.
- Aspiration of the secretions.
- Tracheostomy if needed.

▪ Breathing: is maintained by

- Mouth to mouth breathing
- O₂ mask

▪ Circulation:

- Stop any external bleeding by compression.
- Cannula & saline if possible.

▪ Drugs:

- Morphia 10 mg Slowly IV: to relieve pain "neurogenic shock".
- **3 Anti:** antibiotics, anti-gas gangrene & anti-tetanic serum.

- Morphia is contraindicated in head injuries, unconscious patients and those with respiratory depression.

- Elevate legs & head down to restore cerebral blood flow (Trendlenburg's position).

- Fractures should be immobilized.

- Give sufficient warming (But avoid over-warmth).

➤ At hospital (Better in ICU)

1. Stop hemorrhage: (position - pressure - packing).

2. Resuscitation:

- 2 Cannulas: 1 for fluids "IV line" & the other for sampling (if no veins available (all are collapsed) → venous cut down, open at medial malleolus → saphenous vein)
- O₂ inhalation: to ↑ O₂ content of blood.
- Ryle: to evacuate the stomach for surgery if needed.
- Catheter: monitor urine output.

3. Fluid administration:

○ Crystalloids:

- ▶ In hemorrhage, we start with crystalloids until blood typing & cross matching is done.
- ▶ Types:
 - Normal saline (d.t. equivalent loss of both H₂O & electrolytes).
 - Ringer → Gives K⁺ supply (after ensuring good urine output) in prolonged shock
 - Lactated Ringer's solution → also a buffer, in prolonged shock & acidosis.

- Crystalloids may be the only fluids given to patients with hypovolemia caused by loss of water & electrolytes.
- **Glucose 5% is not used in shock** because glucose is rapidly redistributed, so ↑ amount of ECF (without electrolytes) → dilutional hyponatremia → ↑ ICF (brain edema & ↑ ICT)

▶ Advantages:

- Easy storage & rapid use.
- Doesn't need cross matching.

▶ Disadvantages:

- Diffuse rapidly to extravascular space.

○ Colloids:

▶ Types:

- Plasma substitutes → needs matching & adequate storage.
- Dextran (Polysaccharide polymers):

▪ Types of Dextran:

- High molecular weight dextran "70,000"
- Low molecular weight dextran "40,000"

▪ Disadvantages of dextran:

- Coats RBCs → interferes with blood typing (so blood sample for matching should be taken before giving dextran).
- Coats platelets → interferes with coagulation.
- Antigenic esp. HMW (don't give dextran > 1.5 U)

○ Blood:

- ▶ Given in hemorrhagic shock, when Hct <30 %.

- In the absence of whole blood many substances may be used as human plasma albumin, dextran and artificial blood substitutes.

4. Monitoring: (patient should be continuously monitored to check adequacy of volume replacement)

- Routine:
 - o Pulse & ECG.
 - o Blood pressure.
 - o Temperature.
 - o Urine output (optimum urinary output = 0.5-1 ml/Kg/hr).
 - o ABGs (normal PO_2 =80-100mmHg ,normal PCO_2 =35-45mmHg)
- Others (in severe shock):
 - o **Central Venous Pressure catheter:**
 - ▶ Introduced through an arm vein or Internal Jugular vein or subclavian vein.
 - ▶ Characters:
 - Connected to saline manometer.
 - Monitor CVP for volume replacement.
 - Good indicator for circulatory overload.
 - Normal CVP is 5 cm H_2O .
 - If >15 cm H_2O → Overloading.
 - ▶ Complications:
 - Hemothorax or pneumothorax. – Thrombosis.
 - Pleural injury (should be followed by X-ray to exclude complication).
 - Air embolism. – Sepsis.
 - Arrhythmia.
 - o **Arterial line:**
 - ▶ Monitor ABP.
 - ▶ Repeated ABG assessment.
 - o **Swan Ganz Catheter:** may be needed to measure pressure in pulmonary artery (wedged pulmonary pressure) for early detection of left sided heart failure.

5. Pharmacological support:

- **Inotropic drugs: (Dopamine & dobutamine [selective on β_2])**
 - o To improve cardiac function & O_2 transport.
 - o Dopamine also ↑ renal blood flow.
- **Vasodilators: (Chlorpromazine)**
 - o Given when vascular volume & CVP have been restored to normal
 - o Act by ↓ vascular resistance → ↓ work of the heart & C.O.P.

▪ Vasopressors shouldn't be used as they may aggravate shock in microcirculation beds by increasing pre-existing VC.

➤ Treatment of the cause:

- **Injured great blood vessel:** Surgical exploration & repair.
- **Burns:** (see plastic surgery)
 - Fluids calculated by Evan's or Parkland's formulae.
 - Local wound care.

➤ Treatment of complications:

- **DIC** → Fresh frozen plasma.

➤ Secondary survey: after stabilization, to detect any residual complication.

Failure to respond to fluid resuscitation:**Check C.V.P:**

- ➔ **C.V.P is low:** Continuous bleeding → the causes of massive bleeding include intrapleural – intraperitoneal- retroperitoneal and peripheral bleeding
- ➔ **C.V.P is high:** Tension pneumothorax – cardiac tamponade – cardiac contusion

Irreversible shock

❖ **At some stage hypovolemic shock become refractory to treatment because:**

1. Unsuspected blood in chest & abdomen.
2. Inadequate volume replacement.
3. Multisystem trauma with occult thoracic injury.
4. Acute myocardial infarction, either from direct injury or 2ry to prolonged coronary hypoperfusion.

2- Septic Shock "Endotoxic Shock"**Etiology**

- **It's the most serious type of shock & the most difficult to treat.**

Causative organisms

- Gram –ve bacilli **"The COMMONEST"**.
- Staphylococci.
- Candida.

Source of infection

- **Peritonitis** : caused by perforated viscus , gangrenous bowel or leaking anastomosis.
- **Cholangitis or genitourinary infections.**
- **Infected central venous catheter.**

- **Etiology:** G-ve bacilli, ↓ immunity
- **C/P:** 2 STAGES: warm, cold
- **Comp.:** MOF, DIC, stress ulcers
- **Invest.:** as ATLS + for the cause
- **TTT:** -Resuscitation (organ support)
-Fighting infection (antibiotics)
-Cont. monitoring

Predisposing factors (low immunity)

- Extremes of age.
- Malignancy, malnutrition.
- Chemotherapy, corticosteroids or immunosuppressants.
- Diabetes mellitus.

■ **The incidence of septicemia and septic shock are rising which may be attributed to:**

- i. Developing reservoirs of resistant and virulent organisms.
- ii. Concentration of infected patients in Critical care units.
- iii. More extensive operations in elderly and poor risk patients.
- iv. Salvage of severely injured patients.
- v. Immunosuppression by organ transplantation and chemotherapy.

Pathophysiology

Bacterial endotoxins → $\oplus$ → MQ → $\xrightarrow[\text{TNF, IL2}]{\text{release cytokines}}$ → damage capillary endothelium → edema → VD

(rapid shifting of bl. bypassing capillaries) → tissue ischemia.

(So, septic shock starts as maldistribution of blood followed by myocardial depression & hypovolemia develops).

Clinical Picture

➤ Clinical picture of septic shock:

- **Hyper-dynamic "warm" septic shock (Early phase):** Diagnosis is Difficult and high index of suspicion is required to detect it at early stage.

- Restless & confusion.
- Skin: flushed, warm & dry.
- *Vital data:*
 - Fever $>38^{\circ}\text{C}$ + Chills.
 - Mild decrease in ABP.
 - Tachycardia.
 - Tachypnea.
- High cardiac output.

- **Hypo-dynamic "cold" septic shock (Late phase):**

(Picture similar to hypovolemic shock with reduced cardiac output).

- Skin: cold clammy.
- *Vital data:*
 - SBP $< 90\text{mmHg}$
 - Tachycardia.
 - Tachypnea.
- Oliguria.
- Multi-organ failure starts at this stage.

➤ Clinical picture of the cause:

- UTI, chest infections, peritonitis.....etc.

SIRS

- Is a syndrome in which the septic shock is a part.
- **Etiology:** major trauma, major burn, acute pancreatitis, neglected hypovolemia.
- **Pathogenesis:**
Tissue injury $\rightarrow$ Hypovolemia $\rightarrow$ splanchnic vasoconstriction $\rightarrow$ failure of gut barrier $\rightarrow$ endotoxin and bacterial transfer $\rightarrow$ activation of macrophages and kupffer cells $\rightarrow$ systemic cytokine release $\rightarrow$ sepsis syndrome and multiorgan failure.
- Criteria for diagnosis:
 1. Temp. $> 38^{\circ}\text{C}$ or $< 36^{\circ}\text{C}$.
 2. HR > 90 beat/min.
 3. RR $>$ breath/min.
 4. WBCs > 12000 or $< 4000 / \text{mm}^3$

Criteria for diagnosis

- To diagnose septic shock, the following **two criteria** must be met:
 1. Evidence of infection, through a positive blood culture.
 2. Refractory hypotension (despite adequate fluid resuscitation & cardiac output).
- In addition to the two criteria above, **two or more** of the following must be met:
 1. Hyperventilation (high respiratory rate) > 20 breaths per minute.
 2. ABGs: $\text{PaCO}_2 < 32 \text{ mmHg}$.
 3. WBC count $< 4000 \text{ cells/mm}^3$ or $> 12000 \text{ cells/mm}$.

Complications & causes of death: (80% mortality)

- **Multi-organ failure:**
 - ARDS.
 - Acute renal failure.
 - Hepatic dysfunction.
- **DIC.**
- Acute erosive gastritis "stress ulcer".

Investigations

- **Assessment of general condition:** (should be done serially for follow up)
 - CBC: Marked leucocytosis (or leucopenia, late) & thrombocytopenia (degree of infection & associated complications).
 - ABG: PO₂, PCO₂, pH (hypoxia & hypercapnia in ARDS).
 - Electrolytes & Blood sugar (for dehydration).
- **For the cause & source:**
 - *Isolation of organisms from source of infection & blood by:*
 - Cultures: should be done on anaerobic & aerobic media.
 - Blood cultures.
 - *Location of septic focus:*
 - X-Ray: Abdomen & Chest.
 - U/S & CT scan.
- **For complications:**
 - KFTs & LFTs. (MOF)
 - ECG monitoring. (IHDs)
 - Coagulation profile. (DIC)

Treatment

Admission to ICU as early as possible

➤ **Resuscitation:**

a- **Circulatory support:**

- 1- Fluid replacement: till CVP is 10-12 cm H₂O
 - By Ringer's Lactate + Plasma.
 - If low Hct. → packed RBCs or whole blood transfusion.
- 2- Drugs:
 - Inotropes & vasopressors: dopamine & dobutamine.
 - Given if the patient remains hypotensive despite adequate fluid replacement.

b- **Respiratory support:**

- O₂ by mask.
- If pO₂ < 60 mm Hg → mechanical ventilation.

c- **Renal support:**

- Adequate circulatory support improves renal blood flow.
- Hemodialysis is required in acute renal failure, until the kidneys recover.

d- **DIC:** Fresh frozen plasma.

➤ **Fighting infection:**

a- **Eradication of sepsis:** e. g.

- Drainage of huge abscess or peritonitis.
- Resection of gangrenous bowel.

b- **Antibiotics:**

- Parenteral, combined, broad spectrum started early without waiting for culture & sensitivity results (3rd generation cephalosporin + Amnioglycosides + Metronidazole).
- Then changed according to culture & sensitivity.

➤ **Continuous monitoring:**

- 1- Vital signs (temperature, pulse, BP and RR) and ECG.
- 2- Urine output.
- 3- ABGs, repeated blood culture, CBC, coagulation profile & organ profile.
- 4- CVP, arterial line and pulmonary wedge pressure.
- 5- Strict control of blood sugar has been proved to increase survival.
- 6- Prophylaxis against DVT and stress ulcers.

- **Corticosteroids(A point of controversy):**

- Some say that it can be administered to stabilize cell membrane & antagonize inflammatory mediators.
- Others say that it's not indicated in treatment of septic shock.

E.coli:

- Normal intestinal flora.
- Commonest organism in gram -ve septicemia.
- Commonest organism in abdominal infection.

Prognosis

- High mortality ranges from 25 % up to 90% due to late diagnosis & late treatment.

3- Other Types of Shock

A. Cardiogenic Shock

Definition

- Inadequate blood flow to vital organs due to inadequate COP despite of normal blood volume.

Causes

- Acute myocardial infarction "The COMMONEST"
- Severe arrhythmias.
- Massive pulmonary embolism.
- Cardiac tamponade.
- Myocarditis.
- High spinal anesthesia.

Clinical Picture

(Same as hypovolemic shock +)

- Of the cause: Chest pain.
- Characterized by **congested neck veins & High CVP**.

Treatment

- O₂ Inhalation
- Of the cause
 - M.I. → early thrombolytic therapy & potent analgesics
 - Control arrhythmias.
 - Relief of cardiac tamponade by emergency insertion of cannula to drain blood in the pericardium followed by definitive surgical haemostasis).
 - Mechanical supports:
 - The intra-aortic ballon device serve the following:
 - ☞ Elevating diastolic blood pressure → filling of coronary arteries.
 - ☞ Reduction of myocardial work.

B. Distributive shock

a- Neurogenic sympathetic (Spinal shock)

- **Causes:**
 - High spinal anesthesia.
 - High transection of spinal cord due to fracture spine.
- **Clinical Picture:** (sudden extreme vasodilatation)
 - **Bradycardia + warm skin + ↓ ABP...** etc.
- **Treatment:** Vasopressors + IV fluid therapy

b- Neurogenic vasovagal attack

- **Causes:**
 - Severe pain (Trauma to a trigger area) - Excitement.
- **Clinical Picture:**
 - Bradycardia + Syncope + ↓ ABP.....etc.
- **Treatment:**
 - Trendelenburg's position.

Atropine prevents this reflex but doesn't treat it.

c- Anaphylactic shock**Causes**

- May follow administration of penicillin, sera or dextrans.
- Antigen-antibody "IgE" reaction leads to release of histamine that leads to :
 - Bronchospasm.
 - Laryngeal edema.
 - Massive vasodilatation.

Treatment

- IV crystalloid infusion.
- **IV hydrocortisone** (the most important).
- Antihistaminics.
- Endotracheal intubation.

d- Endocrinal shock**Causes**

- **Adreno-medullary insufficiency:**
 - After adrenalectomy for pheochromocytoma without pre-operative preparation by α & β blockers → sudden withdrawal of high level of catecholamines from the blood.
- **Adreno-cortical insufficiency:**
 - After bilateral adrenalectomy.
 - After removal of cortical tumors
 - After operation on a patient who has had cortisone therapy.
 - Those with Addison's disease, under stress, infections, operations → Addisonian crisis. So, those patients should be nursed in ICU.

Treatment

- Saline infusion.
- IV Hydrocortisone.

Infusion Therapy

Indications

- if the oral route cannot be used or it cannot cope with the normal body requirement either :
 - To correct pre existing deficits.
 - To give the normal daily requirement.
 - To replace losses.

Precautions

1. Clinical observation and assessment.
2. Accurate clear fluid chart for the intake and output daily.
3. Body wt. changes.

4. Check blood chemistry for plasma level of :-

- Na^+ 134-145 m mol/L
- K^+ 3.4 – 4.7 m mol/L
- Cl^- 96 – 106 m mol/L
- Urea 1-7 m mol /L.

Complications

1. Circulatory over load .The use of C.V.P line should prevent this hazard.
2. Pyrogenic reaction
3. Biochemical disturbances.
4. Hypoproteinaemia
5. From the cannula as :
 - Thrombophlebitis.
 - Sepsis.
 - Air embolism.

Solutions for I.V. therapy**A- Crystalloid**

- ▶ Diffuse rapidly from the circulation.
 - Normal Saline contains 0.9% sodium chloride i.e. 153 mmol sodium per liter. – It is isotonic.
 - Dextrose 5% contains 200 calories per liter.
 - Dextrose-Saline contains 4.3 dextrose with 0.18% sodium chloride i.e. one fifth of the amount of sodium in normal saline.
 - Ringers lactate contains :
 - Sodium --- 130 m mol/L
 - Potassium --- 4 m mol /L
 - Chloride --- 110 m mol/L
 - Lactates --- 28 m mol/L

B- Colloids

- ▶ Due to large molecular weight is retained in the circulation.
 - Plasma
 - Dextrane a polysaccharide with two different molecular weights high (70 000) and low (40000).
 - It reduces the blood viscosity and improves the microcirculation.
 - It interferes with coagulation and blood typing so take blood sample for grouping before start the infusion.

ASK YOUR SELF ALWAYS

- 1- How much fluid is needed?
- 2- Which solution should be used?
- 3- What are the possible complications?

ARDS

➡ **See cardiothoracic book.**

CHAPTER: 8

PRE-OPERATION & ANESTHESIA & POST-OPERATION

Pre-operation

Assessment

Objectives of pre-operative assessment

1. Improve the doctor-patient relationship.
2. Reaching **diagnosis** and making **decision**.
3. **Evaluate** the medical condition.
4. **Educate** the patient about the procedure.

Steps of pre-operative assessment

Assessment is done in 3 phases:

- I. Outpatient clinic
- II. Hospital admission
- III. Pre-operative round

History taking

When taking the history, the following should be checked:

- A. **Cardiovascular:**
Anemia, hypertension, IHD.
- B. **Respiratory**
Respiratory infection → should be treated first.
- C. **Gastrointestinal**
Peptic ulcer and GERD.
- D. **Coagulopathy** should be corrected before surgery.
- E. **Genitourinary**
Urinary tract infection.
- F. **Neurological:** Epilepsy.
- G. **Endocrine/metabolic**
Diabetes mellitus (should be properly controlled before elective surgeries).
- H. **Infectious diseases:** HIV, hepatitis and TB.
- I. **Previous surgery:** Type of anesthetic and any problems encountered (either with the patient or one of his relatives).
- J. **Social history**
Patient's occupation.
- K. **Drug history:** Illegal drug abuse.

Physical examination

This will include:

- | | |
|------------------------|---------------------|
| A. General examination | D. Gastrointestinal |
| B. Cardiovascular | E. Genitourinary |
| C. Respiratory system | F. Neurological |

Investigations

(Apart from routine investigations, others are done only when clinically indicated)

- A. **Routine investigations**
 1. Urine analysis, CBC, coagulation profile (BT, PT, PTT).
 2. ABGs, pH and electrolytes (Na^+ , K^+ , Cl^-) and Fasting blood sugar (FBS).
 3. Kidney function tests (KFTs), Liver function tests (LFTs).
- B. **Cardiovascular:**
ECG, echo, ejection fraction.

C. Respiratory:

Chest X-Ray, screening for lung tumors, FEV₁.

D. Endocrine

Pre-operative Preparation

1- **Consent:** must be signed before the operation.

2- **Preoperative round:**

- Final re-examination of the patient.
- Marking the side to be operated upon (in cases of unilateral conditions).
- Making sure that investigations are completed.
- Preparation of blood if transfusion is needed.

3- **Diet:**

- High caloric diet should be taken on the day before the operation.
- **Special preparation in large bowel surgery** → low residue diet.
- The patient should be fasting at least 6-8 hours before operation.

4- **Bowel preparation:**

- Purgatives are no more advisable.
- Soap and water enema may be given in rectal and major operations.
- **In GIT surgeries** → NGT is inserted (better after induction of anesthesia).

5- **Skin preparation:**

- On the morning of operation, shower with 10% povidine-iodine.
- The skin of the operation is shaved, scrubbed with soap and water and antiseptic, and then covered with a sterile dressing.

6- **Maintenance of fluid balance:**

- IV infusion is started in theatre.
- Urinary catheter may be needed to monitor urine output (inserted after the patient is anesthetized).

7- **Pre-anesthetic medications:**

- ⇒ **Timing:** usually given 0.5-2 hours before the induction of anesthesia.
- ⇒ **Selection is largely subjective:** not every patient needs all these medications.
- ⇒ **Goals:**
 1. Sedation/anxiolysis.
 2. Analgesia.
 3. Reduce airway secretions/heart rate control.
 4. Prevent bronchospasm.
 5. Hemodynamic stability.
 6. Prevent and/or minimize the impact of aspiration.
 7. Decrease post-op nausea/vomiting.
 8. Antibiotic prophylaxis.

Sedation

- By **benzodiazepines or opioids (or both)**

Analgesia

- I.M. Morphine 0.05 - 0.15 mg/kg.
- **Side effects:**
 - Nausea
 - Decrease gastric motility,
 - Biliary spasm
 - Respiratory depression especially with benzodiazepines

Airway secretions/Heart rate & prevention of bronchospasm

- **Anticholinergics:** for reduction of secretions.
- **Glycopyrrolate:** β_2 agonist for bronchospasm.

Hemodynamic stability

- Anti-hypertensives.
- For urgent or emergent surgery in patients with poorly controlled hypertension Labetalol is used.

Decrease gastric acidity

- H2 antagonists, PPIs.

Nausea / Vomiting

- Ondansetron.

Medications to be continued

- **Cardiovascular medication:**
calcium-channel blockers, beta blockers, nitrates, and anti-arrhythmic.
- **Bronchodilator**
Should be continued, as withdrawal can precipitate bronchospasm.
- **Antiepileptic treatment:**
- **Steroid intake:**
Sudden withdrawal causes Addisonian crisis
- **Aspirin**

Medications to be stopped

- **Anticoagulants**
 - Warfarin should be stopped at least 4 days before surgery
 - In patients requiring DVT prophylaxis, spinal or epidural can be performed 6 hours after subcutaneous injection of unfractionated heparin and 12 hours after low molecular weight heparin.
- **Contraceptive pill and anesthesia Synthetic estrogens**
- **Monoamine oxidase inhibitors**
- **Selective serotonin reuptake inhibitors (SSRIs).**

Surgery in High Risk Patients

➡ See aid to post-graduate book

Surgery in Diabetes Mellitus

➡ See aid to post-graduate book

Some Anesthetic Considerations for Patients with Medical Problems

➡ See aid to post-graduate book

Anesthesia

Definition

- It is the controlled and reversible depression of the normal response of the whole or part of the body by chemical agents called anesthetics.

Types of Anesthesia

I. General

II. Local (Regional)

A. Central neuro-axial block

- Spinal
- Epidural

B. Peripheral sensory nerve block

- Surface (topical) analgesia
- Local infiltration
- Nerve block
- Local intravenous analgesia

I. General Anesthesia

Definition

- Controlled and reversible depression of the functional activity of the CNS.

Triad of general anesthesia

- Unconsciousness (hypnosis).
- Analgesia.
- Muscle relaxation.

Anesthesia Tools (Drugs & tools)

A. Categories of anesthesia drugs:

- Intravascular induction agents:** to provide rapid smooth induction of hypnosis e.g. barbiturates – non barbiturates.
- Gases and volatile liquids:** given by inhalation to diffuse rapidly from the lungs to the brain e.g. nitrous oxide, halothane, isoflurane.
- Analgesics:** include opioid (as morphine or its synthetic derivatives as pethidine) and non-opioid analgesics as NSAIDs.
- Muscle relaxants and their antagonists:** muscle relaxants act at the neuromuscular junction (NMJ) by blocking the binding of acetylcholine to nicotinic acetylcholine receptors.

B. Anesthesia equipment:

- Airway equipment:** patency of airway should be ensured by oral airways, endotracheal tubes or laryngeal mask.
- Anesthesia machine:** the main components of anesthesia machine are a flowmeter to adjust flow of gases and vaporizers for administration of volatile anesthetics.
- Anesthesia ventilator.**

4. Monitoring during anesthesia:**a. Vital data:**

- Pulse (ECG) and blood pressure.
- Temperature.
- Respiratory monitoring (oxygen saturation).

b. Neuromuscular monitoring: by a device that stimulates a peripheral nerve and records the response of the muscles supplied by it.

Phases of general anesthesia**A. Induction:**

- Intravenous is the most commonly used.
- Recently → inhalational, intramuscular and **rectal**.

It includes:

- A. Induction.
- B. Maintenance.
- C. Recovery.

B. Maintenance:

1. Inhalational anesthetics (recently → intravenous maintenance can be used = total intravenous anesthesia, TIVA e.g. if surgery on airway).
2. Muscle relaxants.
3. Additional doses of narcotics.
4. Mechanical ventilation of paralyzed patients.
5. Close monitoring of vital data.

C. Recovery:

1. Discontinuation of inhalational anesthetics.
2. Antagonism of muscle relaxants by neostigmine with atropine.
3. Discontinuation of mechanical ventilation.
4. Oro-pharyngeal suction and removal of endotracheal tube.
5. Transfer the patient to the **post anesthesia care unit (PACU)** or **surgical intensive care unit (SICU)**.

Complications of general anesthesia**A. Complications during induction of anesthesia:**

1. **Airway manipulation:** e.g. failure to intubate – undetected esophageal intubation – laryngospasm – reflex bronchospasm aspiration of gastric content.
2. **Stress response to intubation:** e.g. hypertension – tachycardia – arrhythmias.
3. **Administration of intravenous drug:** e.g. allergic drug reaction – histamine release leading to bronchospasm and hypotension – injection of wrong drug – awareness during anesthesia – drug overdose leading to hypoventilation and hypotension.

B. Complications during maintenance of anesthesia:

1. Inadequate fluid or blood replacement.
2. Nerve injury due to inappropriate patient position.
3. Undetected patient ventilator disconnection.
4. Corneal ulcer due lack of protective reflexes.
5. Malignant hyperthermia which is a rare but very serious complication.

C. Complications during recovery from anesthesia:

1. Post-operative nausea and vomiting.
2. Sore throat from endotracheal tube replacement.
3. Inadequate recovery of muscle power.

D. Delayed complications of anesthesia:

1. Muscle pain due to fasciculations caused by succinyl choline.
2. Halothane induced hepatotoxicity.

How to minimize complications associated with anesthesia?

1. Proper pre – operative evaluation and preparation.
2. Before induction of anesthesia, the anesthetist must:
 - Check the equipment to be used.
 - Prepare all drugs including emergency drugs.
3. Proper monitoring of the patient intra-operatively.
4. Call for help early when needed.

II. Central Regional Anesthesia

	Spinal	Epidural
<u>Site of injection</u>	Subarachnoid directly into the CSF (intra-theal)	Between dura matter and periosteal lining of vertebral canal
<u>Onset</u>	Rapid (within 5 min.)	Slow
<u>Duration of action</u>	Short acting	Adjustable (according to the dose)
<u>Advantages</u>	1. Rapid onset of action 2. Adequate analgesia	1. Slow sympathetic block → controlled hypotension → less blood loss 2. Multiple shots can be given → adjust the effect and duration 3. Can be given at various levels of the vertebral column
<u>Disadvantages & complications</u>	1. <u>Rapid sympathetic block</u>: • Severe hypotension & bradycardia (→ add IV fluids ± vasopressors) • Ventilator failure (→ ventilator) → to minimize → inject below T10 2. <u>Single shot</u> (can't be repeated → short acting → add epinephrine to prolong) 3. <u>Post-Dural-Puncture Headache (PDPH)</u> → now minimized by using special needles	1. <u>Urinary retention</u> → use catheter ± prophylactic α-blocker 2. <u>Inadequate analgesia (if used alone)</u> → add opioid analgesics N.B. opioid analgesic → respiratory depression in the 1st 24 hours (→ monitoring)

Indications

- 1) Operations below the umbilicus as inguinal hernia, anal operations, operations on the urinary bladder or lower limb amputations.
- 2) In patients with respiratory tract infection.
- 3) In presence of renal or hepatic problems.
- 4) In emergency situations when the patient has a full stomach.

Advantages

- 1) It is simple and easy to be performed.
- 2) It gives adequate muscular relaxation.
- 3) It has minimal interference with cardiac, hepatic or renal functions.

Contraindications

A. Absolute

- 1) Patient refusal.
- 2) Known allergy to local anesthetic drugs.
- 3) Infection or local sepsis at the site of needle insertion (lumbar region).
- 4) Bleeding or coagulation disorders (or patients receiving anticoagulants).

B. Relative

- 1) Hypovolemia.
- 2) Low fixed cardiac output.
- 3) Systemic sepsis.
- 4) Deformities of the spine as kyphosis.
- 5) Neurological or psychological disturbances for medico-legal causes.

III. Local Regional Analgesia

Types

1. Surface analgesia:

- By local anesthetic spray, or cream.
- It is used for mucous membranes as the mouth.

2. Local infiltration analgesia.

- By local injection of the anesthetic in the subcutaneous tissue to block the sensation in a localized area

3. Nerve block.

- Local injection of the anesthetic in the neural sheath around a peripheral nerve.

4. Local intravenous analgesia.

- It is used in the extremities after application of a tourniquet.

Indications

- Minor surgical procedures.
- When general anesthesia is contraindicated e.g. in patients with compromised cardio-respiratory function.

Advantages

- Effects of the drug are limited to the part of body to be operated upon.

Relative contraindications

- In children under 10 years.
- In psychotics.
- Non-cooperative patients.

Post-operative Pain Management

Factors affecting response to analgesia

- Site of operation.
- Age and sex.
- Psychological factors.
- Anesthetic management.
- Bodyweight.

Non-opioid agents

- Used for mild to moderate pain
- Drugs
 - Paracetamol
 - NSAID's
 - Ketorolac IV/IM
 - Diclofenac PO/IM/PR
 - Compound analgesics: paracetamol/codeine

Opioid agents

- Properties:
 - Analgesia.
 - Cough suppression.
 - Histamine release (avoid in asthmatics).
 - Nausea & vomiting.
 - Biliary spasm (avoid in biliary colic).
 - Tolerance.
 - Ventilatory depression.
 - Vasodilatation.
 - Constipation.
 - Pupillary constriction (avoid in head injury).
 - Urine retention (avoid in renal colic).
 - Dependence.
- Drugs:
 - Morphine.
 - Pethidine.
 - Tramadol.
 - Fentanyl.
- Types of administration:
 - Conventional intermittent - morphine 10 mg IM 2-4 hourly.
 - Parenteral routes: - Bolus. - Continuous infusion – rate 3-5 mg/hr.
 - Non-parenteral routes:
 - Sublingual – buprenorphine.
 - Oral.
 - Rectal.
 - Transdermal – fentanyl.

Pain control using local anesthetics

- Local infiltration of site.
- Spinal.
- Epidural.
- Extradural.
- Regional blockage.

Other methods

- Transcutaneous electrical stimulation.
- Cryotherapy.
- Acupuncture.

About morphine

- 1- Avoid it in biliary operations.
- 2- Avoid it in coma, CNS problems, respiratory depression.
- 3- Shouldn't be repeated → addiction.
- 4- Withdrawal side effects:
 - Agitation.
 - Vomiting.
 - Diarrhea.
 - Agrenion.

Post-Operation

1- Postoperative Care

➡ See aid post-graduate book.

2- Postoperative Complications

GENERAL COMPLICATIONS:

- **Cardiac complications:** hypertension, arrhythmias, MI & heart failure.
- **Respiratory complications:** postoperative atelectasis, bronchitis, bronchopneumonia, ARDS.
- **DVT & pulmonary embolism.**
- Nausea & vomiting.
- **Thrombophlebitis of a peripheral or central venous line.**
- **Fever:** may be due to:
 - Reaction to surgery: may occur in the 1st 24hrs.
 - Pulmonary complications (usually the cause in the 1st few days).
 - Surgical site infection: usually in the 4th or 5th day, but it may be delayed.
 - Thrombophlebitis of a peripheral or a central venous line.
 - DVT & pulmonary embolism.
 - UTI.
 - After abdominal surgery (leakage of an anastomosis or development of intra-abdominal abscess).
- **Neurological complications:** stroke or transient ischemic attacks.

LOCAL COMPLICATIONS:

- Seroma.
- Hematoma.
- Wound dehiscence.
- Wound infection.
- Hypertrophic scar & keloid.

Complications may follow abdominal surgery:

- Acute gastric dilatation.
- Paralytic ileus
- Adhesive intestinal obstruction.
- Leakage of an intestinal or gastric anastomosis → may lead to peritonitis, intra-abdominal abscess or fecal fistula.
- Intra-abdominal abscess (pelvic, interloop or subphrenic).
- All the previous complications will lead to nausea & vomiting.

Day Case Surgery

➡ See aid post-graduate book.

CHAPTER: 9

ELECTROLYTES AND ACID-BASE IMBALANCE

WATER ABNORMALITIES

Water Depletion = (Pure Dehydration)

➡ See post-graduate book

Water overdose = (Water toxicity)

➡ See post-graduate book

Body Electrolytes

Sodium

- **Def.:** hypo < 130, hyper > 145 mEq/L
- **Etiology:** relative, absolute
- **C/P:** due to changes in ECF, ICF
- **Invest.:** Na⁺, for the cause
- **TTT:** active TTT cause

HYPONATREMIA

Definition

- Decreased serum Na⁺ < 130 mEq/L.

Etiology

- Dilutional (relative) hyponatremia (Commonest):

A. Increased intake:

1. **Pre operative** water enema.
2. **Operative:** trans-urethral resection of prostate (T.U.R.P) syndrome.
3. **Post operative** over infusion of 5% glucose IV (commonest).
4. **Disease:** Neurosis (excessive intake).

B. Decreased output: e.g. renal failure.

- Non-dilutional (absolute) hyponatremia:

A. Decreased intake: dietary.

B. Decreased output:

1. **Increased plasma loss:** burn, hemorrhage...etc.
2. **Increased GIT loss:** vomiting, GIT suction (especially with intestinal obstruction), diarrhea or GIT fistula.
3. **Increased renal loss:**
 - a. Renal disease e.g. salt-losing nephropathy.
 - b. Adrenal disease (insufficiency).
 - c. Diuretic intake (→ regular check-up is needed).

Clinical picture

- ▶ **Early manifestations (mild cases):** → ↓ ECF
 1. **Due to decreased plasma:**
 - ↓ blood pressure, ↑ heart rate, oliguria and empty neck veins.
 2. **Due to decreased interstitial fluid:**
 - Sunken eye, dry tongue and dry skin.
 - ▶ **Advanced cases (acute reduction):** → ↑ ICF (especially the brain)
 - Non specific CNS manifestations ending in seizures and coma.
- N.B.** if dilutional hyponatremia → manifestations of water intoxication.

Investigations

1. Serum Na⁺ → for diagnosis.
2. Investigations for the cause e.g. renal failure.

Treatment

- A. **Active treatment:**
 1. Mild to moderate cases → normal saline infusion (0.9% NaCl).
 2. Severe cases → 5% NaCl.
- B. **Treatment of the cause.**

Prognosis

- Mortality rate ranging from 5 – 50% according to severity of the case.

HYPERNATREMIA

Definition

- Increased serum Na > 145 mEq/L.

Etiology

- A. **Relative hypernatremia:**
 1. Loss of water and sodium with inadequate replacement (commonest).
 2. Inadequate water intake.
- B. **Absolute hypernatremia:**
 1. **Increased input:** post-operative excessive administration of saline.
 2. **Decreased output:**
 - Hyperaldosteronism → 1ry (Conn's syndrome) or 2ry (liver cirrhosis).
 - Cushing's syndrome.

Clinical picture

1. **Due to increased ECF:**
 - a. ↑ Plasma → hypertension, tachycardia and engorged neck veins.
 - b. ↑ Interstitial fluid → edema and weight gain [the only reliable clinical sign]
2. **Due to decreased ICF (especially the brain):**
 - Non-specific CNS manifestations ending in seizures and coma.

N.B. patients with hypotonic losses present with signs of volume loss e.g. hypotension, tachycardia, dry skin and mucosa.

Investigations

1. Serum Na⁺ → for diagnosis.
2. Investigations for the cause.

Treatment

- A. **Active treatment:** sodium-free water (to be correlated with the duration of hypernatremia).
- B. **Treatment of the cause.**

Prognosis

- Most patients survive, but with residual neurological deficit that becomes persistent in up to 30%.

Potassium

- **Def.:** hypo<3.5, hyper>5.3 mEq/L
- **Etiology:** ↑↓intake, ↑↓loss, shift
- **C/P:** muscle, brain, kidney, liver
- **Invest.:** K⁺, ECG, cause
- **TTT:** cause, specific ttt

Hypokalemia

Definition

- Serum K < 3.5 mEq / L

Etiology

1. **Decreased intake:**
 - IV fluid therapy deficient of potassium.
 - Prolonged decrease of dietary intake
2. **Decreased absorption:** malabsorption syndrome
3. **Increased loss:**
 - A. **Renal loss:**
 - Diuresis:
 - Diuretics especially loop diuretics & thiazides
 - diuretic phase of acute tubular necrosis
 - Osmotic diuresis e.g. fanconi syndrome, RTA
 - Conn's syndrome & Cushing's syndrome (hyperaldosteronism).
 - Alkalosis
 - Drugs: e.g. diuretics.
 - Antibiotics e.g. carbenicillin
 - B. **Gastrointestinal loss:**
 1. Diarrhea especially secretory diarrhea (cholera & ulcerative colitis) → **the commonest cause**
 2. Vomiting.
 3. Gastric suction and external GIT fistula.
 4. T. tube.
 5. Intestinal fistula (e.g. in crohn's disease).
 6. K⁺ losing tumor (villous adenoma).
- A. **excessive tapping of ascites**
4. **Intracellular shift:**
 - Alkalosis
 - Insulin therapy
 - Hypokalemic periodic paralysis.

Clinical Picture

- Mild case: asymptomatic
- I- **Clinical picture of the cause.**
- II- **Effect of hypokalemia on:**
 - Smooth muscles → paralytic ileus.
 - Skeletal muscles → hypotonia & hyporeflexia.
 - Cardiac muscle → Arrhythmias especially in digitalized patients & cardiac arrest.
 - Brain → apathy.
 - Kidney → renal tubular damage (hypokalemic nephropathy) (nephrogenic diabetes insipidus).
 - Liver → liver damage especially in patient with liver disease.

Investigations

- 1- **Serum K:**
 - Is reduced (normal: 3.5-5 mEq/L).
- 2- **ECG:** Sagging depression of ST segment.
 - Flat or inverted T wave
 - Prominent U wave
 - Prolonged QT interval
 - Depression of ST segment
- 3- **Investigations for the cause.**

Treatment

1. **Treatment of the cause.**
2. **Potassium replacement:**
 - **Oral** potassium (in mild cases)
 - **IV** potassium should be given slowly in a large vein with continuous ECG monitoring in ICU.
 - Estimation of potassium deficient = (4.5- serum K concentration) X100 in normal pH in adult.
 - **Safe rules for giving K are: (Rule of 40)**
 - Urine output at least 40 ml/hour.
 - No more than 40 mmol added to 1 L fluid.
 - No faster infusion than 40 mmol/Hour.

Hyperkalemia

Definition

- Serum K > 5.3 mEq / L

Etiology

- 1- **Increased intake:**
 - Excessive therapy with IV potassium (**the commonest cause**).
 - Transfusion of stored blood
- 2- **Decreased Excretion:**
 - A. **Renal cause: (the 2nd common cause)**
 - Acute renal failure
 - Severe chronic renal failure
 - Tubular disorder e g type IV RTA
 - B- **Adrenal cause:** Addison's disease
 - C- **Drugs:** Potassium- Sparing diuretics.

3- Extracellular shift:

- Tissue damage:
 - Haemolysis
 - Rhabdomyolysis
 - Tumour lysis syndrome
 - Acidosis
 - Insulin deficiency
 - Hyperkalemic periodic paralysis
 - Drugs e.g. Succinylcholine.

4- Pseudohyperkalemia:

- Polycythaemia
- In vitro haemolysis

Clinical Picture

- Clinical picture of the cause: 4A
 - Asthenia of the skeletal muscles
 - Atonia of the intestinal muscles
 - Arrhythmia of the heart, bradycardia and arrest
 - Apathy

Investigations

1. **Serum K:** is increased (normal 3.5-5 mEq/l).
2. **ECG:**
 - Prolonged P-R interval
 - Loss of P wave
 - Widened QRS complex
 - Bradycardia, heart block, sine wave pattern & arrest

3. Investigation for the cause**Treatment**

1. **Treatment of the cause.**
2. **Correction of hyperkalemia: (BIG C)**
 - If $K > 7$ mEq/L → dialysis.
 - If $K < 7$ mEq/L → IV sodium **B**icarbonate 100 ml IV infusion.
 - 20 units regular **I**nsulin infusion over 3 hours + **G**lucose 25% 100 cc.
 - Beta-2 stimulation by injection or inhalation.
 - IV **C**a gluconate as calcium antagonizes K effect on the heart.

Abnormal metabolism of calcium

I- Hypercalcemia

Definition

- Serum calcium level > 11mg/dl

Etiology

- **Def.:** >11 mg/dl
- **Etiology:** with ↑ or ↓ parathormone
- **C/P:** asymptomatic, bone, stone, groan
- **Invest.:** parathormone, Ca^{+2} , ph^{+3} , comp.
- **TTT:** IV fluids. cause

With normal or elevated parathyroid hormone level

1. 1ry or 3ry hyperparathyroidism
2. Familial hypercalcemia

With low or suppressed parathyroid hormone level

1. Malignancy (lung, breast, renal, anal, colonic, thyroid carcinoma, lymphoma, multiple myeloma)
[commonest cause in hospital]
2. ↑ Vit. D (intoxication, sarcoidosis & HIV)
3. Immobilization
4. Milk alkali syndrome
5. Thyrotoxicosis
6. Thiazide diuretics
7. Addison disease

Clinical picture

➡ Asymptomatic (the commonest presentation)

1- Bone:

- Bone pains.
- Osteitis fibrosa cystica → multiple bone cysts.
- Pathological fractures.

2- Stone:

- Recurrent renal stones.
- Nephrocalcinosis.
- Polyuria.

3- GIT:

- A., N., V.
- Peptic ulcer
- Pancreatitis

4- Psychic (especially in women)

- Apathy
- Depression
- Lack of concentration

Investigations

➡ Should be done in the following order:

1. 1st → measure PTH
 2. 2nd → to determine type → measure Ca^{+2}
 - ↑ in 1ry or 3ry hyperparathyroidism
 - Normal or low in 2ry hyperparathyroidism (normocalcemic hyperparathyroidism)
- Ph^{+3} → decreased
- except, increased with chronic renal failure (2ry hyperparathyroidism)

3. 3rd → to visualize the effect:
 - X-ray spine → multiple bone cyst (osteitis fibrosa cystica)
 - Plain X-ray kidney & IVU → renal stones
 - Upper GI endoscopy → peptic ulcer
 - ECG → short QT interval → arrhythmia
4. Investigations for localization in recurrent cases:
 - CT, U/S, MRI
 - Thallium ²⁰¹, Tc⁹⁹ radioisotope subtraction scan.
 - Sesta MIBI scanning (**The most accurate & better than U/S**)

Treatment

- I. TTT of hypercalcemia (Medical)
 - ➔ **Mainly:**
 - Rehydration with IV fluids
 - Bisphosphonates
 - ➔ **Others:**
 - Calcitonin IV
 - Prednisolone
 - Lasix
 - Hemodialysis
- II. TTT of the cause e.g. hyperparathyroidism

II- Hypocalcemia

➔ **See post-graduate book**

Acid base disorders

I- RESPIRATORY ACIDOSIS

➤ Low PH ($\uparrow$ H⁺) due to respiratory causes

Etiology

- Hypoventilation ($\downarrow$ alveolar ventilation), e.g.:
 - Central cause → CNS depression by injury or drugs (opiates)
 - Peripheral cause:
 - Muscle weakness (myasthenia gravis)
 - Obstructive pulmonary disease, e.g. COPD

Clinical picture

- Cyanosis
- Irritability

Treatment

- Mainly by mechanical ventilation

II- RESPIRATORY ALKALOSIS

Etiology

- Hyperventilation, due to :
 1. Hysterical
 2. Hyperpyrexia

Clinical picture

- $\uparrow$ respiratory rate
 - If prolonged → tetany.
 - If severe → respiratory arrest

Treatment

- Patient respire into a paper bag.

III- METABOLIC ACIDOSIS**Etiology**

1. ↑ Production of acids
 - DKA
 - Lactic acidosis due to sepsis / shock
2. Loss of HCO_3^-
 - Renal failure (acute / chronic)
 - GIT (diarrhea, pancreatic or small intestinal fistula, ureterosigmoidostomy)

Clinical picture

- ↑ rate & depth of breathing (Kusmmaul respiration)

Treatment

- **Mild to moderate cases:**
→ Treatment of the underlying cause.
- **Severe cases:**
→ Sodium bicarbonate should be used for correction of:
 1. Acidosis associated with myocardial depression.
 2. $\text{pH} < 7.2$ or $\text{HCO}_3^- < 15 \text{ mEq/L}$.
- Estimated as: body weight (in Kg) X 0.3 X base deficient

IV- METABOLIC ALKALOSIS**Etiology**

1. Loss of H^+
 - GIT → vomiting & diarrhea (the commonest causes d.t. pyloric obstruction)
 - Kidney → paradoxical aciduria (in case of metabolic alkalosis)
2. Gain in HCO_3^- → NaHCO_3 administration (in TTT of acidosis / milk alkali syndrome)

Clinical picture

1. Of the cause
2. In severe cases → tetany occurs

Treatment

- Treatment of the cause.
- Mild cases → IV saline infusion.
- Severe cases → ammonium chloride IV slowly (if failed above measures).
- If
 - Hypokalemia → IV ringer.
 - Tetany → 10 ml of 10% calcium gluconate IV over 10 min.

Postoperative fluid & electrolyte therapy

➡ In prescribing fluid regimens for postoperative patients, three points are considered:

1. Basal requirements.
2. Pre-existing dehydration, electrolytes loss and acid-base disturbance.
3. Continuing abnormal losses over and above the basal requirements.

➡ The daily requirements of water and electrolytes in an adult are:

- Water 35ml/kg.
- Sodium 1mmol/kg.
- Potassium 1mmol/kg.

➡ The usual daily postoperative fluids for uncomplicated surgery in an adult:

- Three liters of fluids=6 bottles. Add 200ml per day for each 1°C rise in temperature.
- 500 ml saline (0.9% sodium chloride) provides the daily requirements of sodium and chloride.
- The remaining volume requirement (2.5l=5 bottles) is given as 5% dextrose (glucose).
- Potassium supplements are given after 48 hours. Either dextrose is replaced by kadalex which contains 27mmol of potassium/l, or potassium supplements in the form of potassium chloride is added.

CHAPTER: 10

TUMORS IN SURGERY (ONCOLOGY)

- 1- General scheme for malignancy
- 2- Biology of cancer
- 3- Tumor markers
- 4- Radiation therapy
- 5- Cytotoxic chemotherapy

Tumors in Surgery (Oncology)

Definition

- Tumor is autonomous, excessive, purposeless and pathological proliferation of cells that continues indefinitely in absence of physiological stimuli.

General Scheme of Malignant Tumors

Predisposing Factors

1. **Idiopathic.**
2. **Genetic factors:** e.g.

Syndrome	Inheritance	Associated tumors
Familial polyposis coli & Gardner syndrome	Dominant	Colorectal cancer Osteoma of the jaw Desmoid tumor
MEN-I	Dominant	Parathyroid tumors Pancreatic tumors Pituitary tumors
MEN-IIa	Dominant	Medullary carcinoma of thyroid Pheochromocytoma Parathyroid tumor
MEN-IIb	Dominant	Medullary carcinoma of thyroid Pheochromocytoma Neurofibroma
Li-Fraumeni syndrome (P53)	Dominant	Leukemia Osteosarcoma Brain tumors
Familial breast cancer (BRCA-I & BRCA-II)	Dominant	Breast cancer Ovarian cancer Prostate cancer

3. Environmental factors:

- **Chemical:**
 - Tobacco: lung cancer, head and neck cancer and bladder cancer.
 - Alcohol: head and neck cancer, esophageal cancer and hepatoma.
 - Asbestos: mesothelioma of lung.
 - Aflatoxin: hepatoma.
- **Physical:**
 - UV-rays: melanoma and non-melanoma skin cancers.
 - Exposure to irradiation.
 - Mechanical irritation: e.g. gall bladder stones.
- **Infection:**
 - Viral: HPV (cervix and penile cancer), HIV (Kaposi sarcoma, lymphoma and anal cancer) and HBV or HCV (hepatoma).
 - Bacterial: H. pylori (cancer stomach).
 - Parasitic: Bilharziasis (cancer bladder).

4. **Obesity:** associated with tumors in the breast, endometrium, colon & esophagus.

5. Pre-malignant tumors.

Incidence

More common in elderly males

Pathology

- Site: according to type of tumor
- Macroscopic picture: according to site of tumor:-
 - **In solid organs** → star-shaped mass with infiltrating edges + areas of hemorrhage & necrosis.
 - **In hollow organs** → ulcerative, cauliflower mass or infiltrative lesion.
- Microscopic picture: according to lining epithelium of tumor + criteria of malignancy (Pleomorphism & mitotic figures, acidophilic cytoplasm and acidophilic nuclei).
- **Stages of cancer development:**
 1. Hyperplasia
 2. Metaplasia
 3. Dysplasia
 4. In situ carcinoma
 5. Invasion
 6. Metastasis

Spread

- **Direct (Local) spread**: to adjacent tissues.
- **Lymphatic spread**: to draining L.Ns.
- **Blood spread (2L2B)**: liver, lung, bone & brain.
- **Transcoelomic spread**: in GIT malignancies:
 - ⇒ When the tumor reaches the serosa → malignant cells spread toward the peritoneal cavity → peritoneal nodules + malignant ascites → then to pelvis → Krukenburg tumor + nodules in Douglas pouch.

Clinical Picture

Malignant tumors may cause:

- Rapidly progressive system effect.
- Predominant local effect

1. **Local effect of tumors**: varies according to site:
 - a) Tumors on surface of the body → may bleed or discharge.
 - b) Tumors in a hollow viscus or duct → obstruction, bleeding, discharge, perforation.
 - c) Tumors in closed space →
 - Pressure symptoms (e.g. ↑ ICT in brain tumors)
 - Invasion of the organ → affect the function of the organ → organ failure.
 - Invasion of nerves → severe pain.
2. **Systemic effect of tumors**
 - A. **Generally** → cachexia, anemia, weight loss and fever of unknown origin (FUO).
 - B. **Spread**
 - a. Direct: to nearby organs.
 - b. Blood: mainly to :
 - 1) Brain → ↑ ICT, focal neurological manifestations.
 - 2) Lung → infiltration, cavitations, infection and pleural effusion.
 - 3) Bone → bone ache and pathological fractures.
 - 4) Liver → pain in Rt. hypochondrium, jaundice, enlarged hard tender liver.

c. Lymphatic spread:

- 1) Enlargement of regional lymph nodes.
- 2) Later on enlargement of supraclavicular LNs (Troisier's sign).

d. Transcoelomic spread: by seeding or by retrograde lymphatic spread.**C. Some tumors may cause non-metastatic (paramalignant) manifestations**

a. Clubbing & hypertrophic osteoarthropathy.

b. Neurological manifestation:

- Peripheral neuropathy, myopathy.
- Polymyositis, dermatomyositis and myasthenia gravis.

D. Secretion of abnormal proteins may cause characteristic syndromea. Appropriate secretion where the tumor cells secretion is related to tumor site e.g. tumor of adrenal cortex → excess cortisone → Cushing disease.b. Inappropriate secretion not related to site of tumor e.g. Bronchogenic carcinoma → ↑ ACTH → stimulate normal adrenal gland → ↑ cortisone level.**Staging**

- **Stage I** → Tumor is localized to the organ.
- **Stage II** → Tumor with enlarged mobile draining LNs.
- **Stage III** → Tumor with enlarged fixed draining LNs (± local invasion).
- **Stage IV** → Tumor with distant metastasis.

TNM classification of malignant tumor:

It is a cancer staging system that describes the extent of cancer in a patient's body. **T** describes the size of the tumor and whether it has invaded nearby tissue, **N** describes regional lymph nodes that are involved, and **M** describes distant metastasis.

Investigation of malignant tumors▪ **For diagnosis**: according to type of tumor.▪ **For staging**:1. **Sentinel Lymph Node Study**:

- By injection of methylene blue or radioactive isotope → follow-up → till we find the sentinel lymph node.
- Then it is excised and frozen section is done for it to know whether affected by the cancer or not.

2. **Lung** → CXR.3. **Liver** → abdominal ultra sound and liver function tests.4. **Bone** → bone scan (Tc 99).5. **Brain** → CT scan and MRI.▪ **For pre operative preparation**:

- CBC, FBS, LFTs, KFTs.

▪ **For follow up**:1. **Tumor markers**: CA15-3 and CEA.2. **Biochemical investigations (hormonal receptors)**: estrogen and progesterone.▪ **For screening**: as in

1. Mammography in cancer breast

2. Calcitonin level in medullary carcinoma of thyroid.

Treatment

1. Surgery.

- ▣ **Primary tumor:** radical surgery aims at excision of the primary tumor with as wide safety margin.
- ▣ **Lymph nodes:** the treatment of lymph nodes varies from one tumor to another.
- ▣ **Precautions:** during surgery care is taken to avoid spillage of malignant cells.
- ▣ **Advantages:** surgical excision is both quick and effective for the largest number of cures to confirm that a tumor has been fully excised.
- ▣ **Disadvantages:** may produce functional and cosmetic disabilities, it can't be applied if the tumor is fixed to a vital structure or if has produced distant spread.

2. Radiotherapy

- ▣ May replace surgery or may be given in addition.
- ▣ Common indications. Cancer of the larynx so as to preserve the voice, early Hodgkin's disease, early prostate cancer, and as a part of conservative therapy for early breast cancer(after surgery).
- ▣ **Methods:** powerful x ray, gamma rays, electrons, or heavy particles.
- ▣ **Advantages.**
 - a. Can preserve the anatomical structures that surround a cancer.
 - b. Radiation can destroy microscopic extensions of cancerous tissue around a tumor.
 - c. Is a safer option for older, frailer patients who might have difficulty recovering from surgery.
 - d. Patients treated with radiation usually don't require hospitalization.
- ▣ **Disadvantages:**
 - i. In general is much less sensitive.
 - ii. Burns of the skin or enteritis.
 - iii. Compared to surgery ,radiotherapy is slower as it usually takes five to eight weeks ,not suitable for metastatic cases .

3. Chemotherapy:

- ▣ **Common indications:** is the main ttt of leukemias.
- ▣ Better results are obtained from combination chemotherapy.
- ▣ **Advantages:** travel through circulation, can reach malignant cells anywhere.
- ▣ **Disadvantages:** often kill many healthy cells and thus bring on serious side effects, tendency to damage to the rapidly growing cells of the bone marrow.

4. Hormone therapy: has relatively mild side effects because its actions are limited largely to tissues with receptors for specific hormones.

5. Immunotherapy:

- ▣ **Non specific:** BCG induce remission in cases of transitional cell carcinoma of the urinary bladder.
- ▣ **Specific:** monoclonal antibodies from a single clone of lymphocytes.
- ▣ **Bone marrow transplantation:** not a therapy in itself but in sometimes used to strengthen the depleted bone marrow (blood-making system).

6. Biological therapy:

- ▣ Tremendous advances in oncology and molecular biology allowed the emergence of newer anti-cancer drugs which antagonize certain biological agents expressed by certain tumors as the next examples:

Drug	The biological agent antagonized
Herceptin: for breast cancer	Her-2 neu receptors.
Gleevec (imatinib): for gastro intestinal stromal tumors, GIST	Tyrosine kinase

	Early cancer (Potentially curable)	Advanced cancer (Incurable)
Definition	stage I or II	stage III or IV
Aim	Cure	Palliation
Disease status	Mainly local disease ± micro-metastasis	Mainly systemic disease
Primary treatment	Surgery ± radio-therapy (Local treatment)	Chemotherapy & endocrinal therapy (Systemic treatment)
Adjuvant treatment (Palliative)	Endocrinal & chemotherapy	Simple mastectomy & radio-therapy have limited role in local control

Tumor Markers

Definition

- Some malignant tumors produce substances (enzyme, hormone, protein or antigen) that can be detected in the blood and may be used as marker of both the presence of the tumor and screening for early recurrence following treatment.
- Tumors markers are also useful in assessing the response of Tumors to treatment.

☞ **An ideal tumor marker should have the following characteristics:**

- Always produced by the tumor.
- Gives a sensitive indication of the tumor mass.
- Produced by recurrent and metastatic disease.
- Specific for the disease and easy to measure.
- The tumor should be treatable.

Clinical use of tumor markers

Marker	Cancer	N.B.
CA 15-3	❖ Breast cancer	
Calcitonin	❖ Medullary carcinoma of thyroid	• If high total thyroidectomy is indicated even if thyroid clinically & by investigations is normal
Thyroglobulin	❖ Follicular & papillary thyroid carcinoma	
Human chorionic gonadotrophin (β-HCG)	❖ In both seminoma & teratoma	• AFP: - Normal level (0-10 mg/dl).

α -Feto-protein (AFP)	❖ Hepatoma (HCC) ❖ Only in teratoma, never in seminoma	- > 500 mg/dl → is highly suggestive of malignancy. - It is non-specific & may rise in: 1. Pregnancy. 2. Liver cirrhosis. 3. Chronic hepatitis. • These 3 markers are used for: 1. Diagnosis 2. Prognosis 3. Follow up after surgery
LDH	❖ In seminoma (60% of cases) ❖ Also, in neuroblastoma	
Carboxy-prothrombin	❖ HCC ❖ Fibrolammelar type of hepatoma.	• More specific
Carcino - Embryonic Antigen (CEA)	❖ Cancer stomach ❖ Cancer pancreas ❖ Cancer Breast ❖ Cancer colon	• > 65% indicates extensive spread
CA19-9	❖ Cancer stomach ❖ Pancreas	
CA 72-4	❖ More recent & specific for cancer stomach	
POFA (pancreatic oncofetal Ag)	❖ Cancer pancreas	
PCA (Pancreatic Cancer Associated Ag)		
5-HIAA (5-OH Indol Acetic Acid in urine)	❖ Cancer appendix	
VMA (Valinyl Mandelic Acid in urine)	❖ Pheochromocytoma	
Nuclear matrix protein 22	❖ Cancer urinary bladder	• For screening

Acid phosphatase	❖ Cancer prostate	
PSA (Prostatic specific Ag)	❖ Cancer prostate	• More specific than acid phosphatase. • It is a serum protease helping in liquefaction of semen. • Normally 0-4 ng/ml. • If > 30 ng/ml → mostly metastatic cancer prostate. • If < 15 ng/ml → may be BPH or cancer prostate.
CA 125	❖ Cancer ovary	
Enolase +ve	❖ Pheochromocytoma. ❖ Neurofibromatosis. ❖ Wilm's tumor.	
KRAS	❖ Recently in most of GIT tumors & cholangiocarcinoma	
Alkaline phosphatase	❖ Osteosarcoma	
PAP (Placental Alkaline Phosphatase)	❖ Cancer ovary. ❖ Seminoma.	

Radiation Therapy

Definition

- It is the treatment of malignant tumors with ionizing radiation, which is composed of high energy rays that cause ejection of atomic electron when absorbed.

Indications

- **Curative:** used alone in some tumors e.g. Hodgkin's disease.
- **Neoadjuvant:** for down-staging of the tumor before surgery.
- **Adjuvant:** for radical removal of tumor residuals after surgery.
- **Palliative:** if the case is inoperable and irresectable.

Mechanism of Radiation Therapy

- The effects are due to production of highly reactive O_2 free radicals.
- Effect is enhanced in the presence of molecular oxygen.
- The radicals interact with molecules in the irradiated cells → change molecular structures.
- Such changes may damage cellular DNA or the protein mechanism needed for cellular reproduction and metabolism.

Radio sensitivity

- Is the susceptibility of a certain cell to be damaged by ionizing radiation rapidly dividing cells tend to be much more radiosensitive than slowly dividing ones.
- **Types:**
 - 1- **Curable:** e.g. rodent ulcer.
 - 2- **Resistance:** e.g. adenocarcinoma.
 - 3- **Sensitive:** e.g. Wilm's tumors.

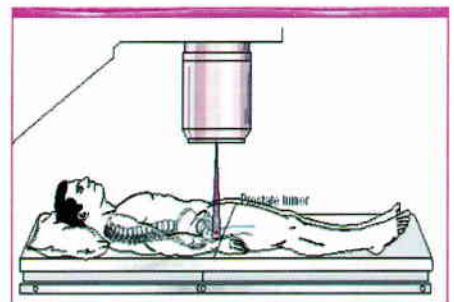
Dose

- Doses are measured in gray (Gy) units, which are equal to the absorption of 1 joule /kg of tissue.
- One gray is equal to 100 rads.
- The probability of controlling a tumor with irradiation is inversely related to the size of the tumor.

Modes of delivery

1. External beam radiation teletherapy (EBRT):

- It is the most common mode of delivery.
- It uses a device located some distance from the patient to focus radiation on the target tissues.
- The goal is to deliver maximal radiation to the tumor while excluding normal surrounding tissue.



2. Brachytherapy:

- e.g. intestinal irrid. needle
- The principle is to place a radiation source near the site of the primary tumor either surgically or through the normal body openings e.g. in prostate cancer.
- Other methods involve the use of catheters that can be filled with radioisotopes.

3. Intra-operative radiation therapy:

- Used in the operating room after tumor resection.

4. Preoperative:

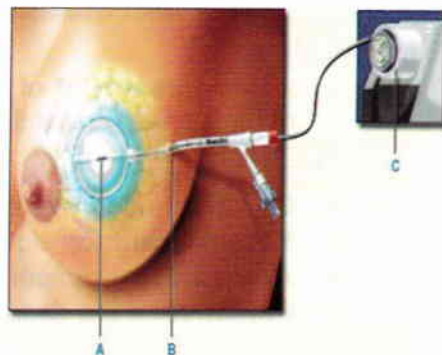
- e.g.in cancer rectum to change it from irresectable cancer into resectable cancer (down staging).

5. Post-operative**6. Systemic targeting:**

- Depends on unique features of the tumor cells, as in the specific uptake of radioactive I^{131} by certain types of thyroid cancers.
- **Advantage** → allows treatment of tumors which may have metastasized or spread beyond anatomic boundaries.

7. Radio-immunotherapy:

- It is an experimental mode of systemic delivery.
- Radioisotopes are bound to antibodies targeting tumor-specific antigens.
- This technique has shown great promise in the treatment of many cancers, including hepatocellular carcinoma and lymphoma.

**Disadvantages**

1. **Resistance** of some tumors to radiotherapy e.g. adenocarcinoma.
2. **Slower onset** of action when compared to surgery.
3. **Associated complications:** end arteritis obliterans, skin burn, ulcers, bone marrow depression, enteritis, intestinal obstruction or fistula formation, sterility, interstitial pulmonary fibrosis and radiation sickness.

Cytotoxic Chemotherapy

Definition

- It is treatment of malignant tumors using pharmacologic agents systemically.
- The ideal tumor drug would kill cancer cells without affecting normal tissues.

Basic principles of combined therapy

1. Use of effective agent.
2. Use agents with different mode of action (synergism).
3. Use agents with non-overlapping toxicity.
4. Consider spatial co-operation.

Indications

- **Curative:** used alone in some tumors e.g. leukemia.
- **Neoadjuvant:** for down-staging or shrinkage of the tumor before surgery.
- **Adjuvant:** for elimination of any microscopic foci of systemic malignancy.
- **Palliative:** if the case is inoperable and irresectable.

Classes of Chemotherapeutic Agents

- Chemotherapeutic drugs are generally classified as:
 - Cell cycle specific (CCS) drugs, which are toxic to actively proliferating cells;
 - Cell cycle-nonspecific (CCNS) drugs, which are capable of killing cells that are not dividing during drug exposure.
- These two classifications are not absolute, and many drugs may overlap between the two categories.
- Combination helps against tumor resistance and increase the tumor killing while avoiding the compounding of toxic effects.

	Mechanism	Uses	Examples
Alkylating agents	Breaking of DNA, miscoding or cross linking	- Hematological tumors - Some solid tumors e.g. breast – lung	Cyclophosphamide Cisplatin
Anti-metabolite	- Incorporate in nucleic acid - Shut down of cellular synthetic mechanism	- Hematological tumors - Some solid tumors e.g. breast – GIT	Methotrexate (MXT) 5-Fluorouracil
Antibiotics	- Blocks DNA synthesis - Breaking DNA strands - Anti-tumor activity	Solid tumors	Doxorubicin Dactinomycin
Plant alkaloid	<u>Vincristine</u> → arrest mitosis in metaphase <u>Others (e.g. Etoposide)</u> → topoisomerase enzyme inhibitors → inhibit cleavage and re-ligation of DNA	- Hematological tumors - Some solid tumors (breast – kidney – testis – head & neck cancers)	Vincristine Etoposide (derived from natural plants)

Side-effects of chemotherapy

- Metallic taste for 2-3 days after treatment.
- Nausea, vomiting, diarrhea.
- Rashes and ulceration of the skin.
- Hair loss (not all drugs cause this).
- Drug resistance** causes chemotherapy failures → combination therapy.
 - ➔ **Mechanisms**
 - Pumping drugs out of tumor cells.
 - Alteration of target enzymes.
 - Increased production of a target enzyme to overcome the drug.

CHAPTER: 11

MISCELLANEOUS SUBJECTS

- 1- Nutrition
- 2- Obesity
- 3- Surgical Hypertension
- 4- Transplantation
- 5- Imaging
- 6- Laser
- 7- Clinical Audit
- 8- Surgical Ethics
- 9- Surgical Drains

Surgical Nutrition

Physiological consideration

- Normal person needs 30 Kcal/kg body weight (average 2100 Kcal/day).
- The energy given by each type of food:
 - 1 gm carbohydrate → 4 Kcal
 - 1 gm protein → 4 Kcal
 - 1 gm fat → 9 Kcal
- Calorie (CHO + fat) : nitrogen = 200 Kcal : 1 gm
- So, the ratio of the different items in a well-balanced diet:
 - carbohydrate → 50%
 - fat → 35%
 - protein → 15%
- Maintaining a healthy nutritional status requires the following daily balanced supplementation:

- **Etiology:** starvation, ↓catabolism
- **Effects:** ↓ (healing, immunity, recovery, tolerance to radio & chemo)
- **Diagnosis:** anthropometric, lab.

	Per Kg	For 70 Kg person
- Water (ml)	35	2450
- Carbohydrate (gm)	2	140
- Fat (gm)	3	210
- Protein (gm)	0.7	50
- Nitrogen (gm)	0.7	7
- Na ⁺ (mmolL)	1	70
- K ⁺ (mmolL)	1	70

- Proteins are better assessed by its nitrogen contents.
- So, if the intake of nitrogen exceeds its amount in urine = (positive nitrogen balance) = anabolic state.
- If the reverse = (negative nitrogen balance) = catabolic state.

Malnutrition in surgical patient

Incidence

- About 30% of patients with GIT diseases.
- It reaches 60% in those with prolonged hospital stay.

Etiology

A. Starvation (↓ input):

1. Social causes. as poverty & neglected elderly
2. Loss of appetite by chronic diseases.
3. Dysphagia.
4. Repeated vomiting.
5. Malabsorption syndrome → in {
 - Chronic pancreatitis
 - Short gut syndrome
 - Crohn's & ulcerative colitis
6. Post-operative restriction of oral intake (e. g. paralytic ileus).

B. Hypercatabolism (↑ output):

1. Major trauma and burns.
2. Major surgical operations.
3. Severe acute pancreatitis.
4. Major sepsis e. g. septicemia.

Effects on the outcome of surgery

➤ **Operative mortality & morbidity ↑ when 20% of the body weight is lost.**

1. Impairment of wound healing. e.g: burst abdomen
2. Immunosuppression with ↑ susceptibility to infection.
3. Delay physical recovery & ↑ hospital stay.
4. Decreased tolerance to radiotherapy and chemotherapy.

Diagnosis

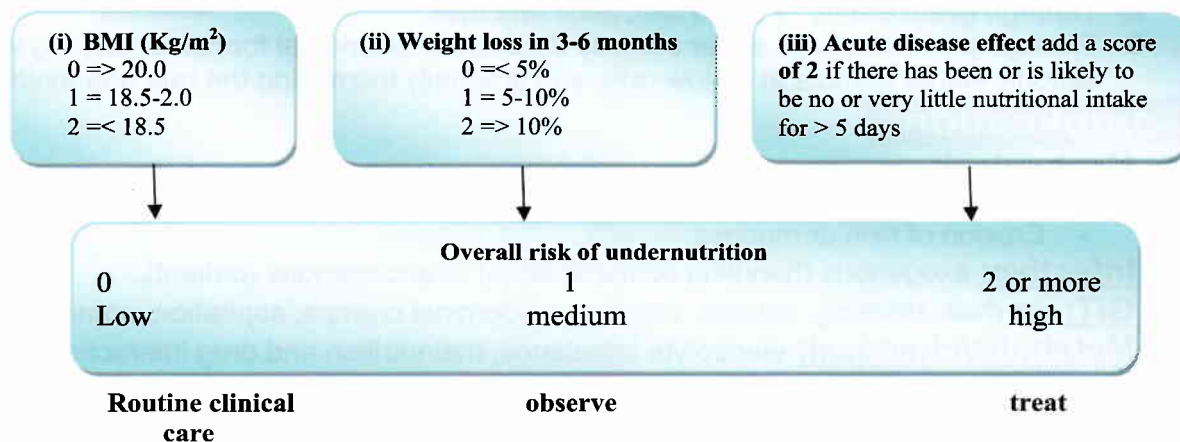
A. Anthropometric measures:

1. Recent un-intentional **weight loss** > 10%.
2. **Body weight** is < 80% of the ideal for height.
3. **Muscle mass:** Mid-arm muscle circumference < 80% of the average.
4. Triceps **skin fold** thickness.

B. Laboratory:

1. Serum albumin < 3.5 gm/L.
2. Determine nitrogen balance (whether +ve / -ve).
Input – output → +ve nitrogen balance (anabolism).
→ -ve nitrogen balance (catabolism).
3. TLC < $1.200 \times 10^9/L$.
4. Impaired hypersensitivity reaction.

The MUST tool: the British association of parenteral and enteral nutrition introduced mal-nutrition universal screening tool (MUST)



Types of Surgical nutrition

A. Enteral nutrition.

B. Parenteral nutrition.

A. Enteral Nutrition

Definition

- Delivery of nutrients into the GIT.

Indications

- Patient in whom oral intake is inadequate whenever there is a functional accessible
- GIT (as comatosed patient, severe dysphagia, neck surgery, low output GIT entrocuteaneous fistula and burns).

Routes of administration

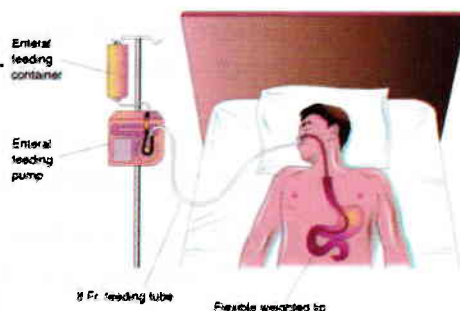
- Sip feeding:** whole food by mouth (fluid formula).
- Tube feeding techniques:**
 - Nasogastric tube (NGT):** Ryle's tube
 - Gastrostomy:** if nasogastric feeding is not possible e. g. upper GIT obstruction.
 - Jejunostomy:** if the stomach is diseased.

Administration formulae

- Through gastrostomy → liquid diet, juice and milk.
- Through jejunostomy → either partially digested or elemental formulae, starting with isotonic sterile formula at a slow rate, and gradually increasing the rate and tonicity.

Complications

- Mechanical:**
 - Malposition, displacement, blockage, breakage or leakage of the tube.
 - Erosion of skin or mucosa.
- Infective:** exogenous (handling contamination) or endogenous (patient).
- GIT:** diarrhea, bloating, nausea, vomiting, abdominal cramps, aspiration, constipation.
- Metabolic/chemical:** electrolyte imbalance, malnutrition and drug interactions.



➤ Tube Care:

- Flushed with water at least twice per day.
- If obstructed with solidified diet → instillation of chemotrypsin inside the tube may salvage a partially obstructed tube.

B. Total Parenteral Nutrition (TPN)

Definition

- Provision of all nutritional requirements through intravenous route without use of GIT.

Indications

- The principle indication for TPN is found in seriously ill patients suffering from protein calorie malnutrition where the use of GIT for feeding is not possible as follow:
 - 1- GIT is **blocked** e.g. stricture, neoplasm or extrinsic mass.
 - 2- GIT is **short** e.g. short gut syndrome.
 - 3- GIT is **fistulated** e.g. enterocutaneous fistula.
 - 4- GIT is **inflamed** e.g. inflammatory bowel disease.
 - 5- GIT can **not cope** e.g. severe trauma or hyper-catabolic state.
 - 6- Radiation enteritis.
 - 7- Moderate to severe pancreatitis.
 - 8- Paralytic ileus.

Route of Administration

- Central:** catheter inserted via subclavian or internal or external jugular vein.
- Peripheral:** (appropriate for short term feeding up to 2 weeks)
 - iv. Peripherally inserted central venous catheter (PICC) line.
 - v. Conventional short cannula in the wrist vein (isotonic fluid).

Calculation of requirements

- Normal person needs 30 Kcal\ Kg body weight, 2400 Kcal/day.
- Surgical patient need 40 Kcal\Kg body weight.
- The energy given by each type of food:
 - 1 gm carbohydrate → 4 Kcal
 - 1 gm protein → 4 Kcal
 - 1 gm fat → 9 Kcal
- Calorie (CHO + fat) : nitrogen = 200 Kcal : 1 gm
- So, the ratio of the different items in a well-balanced diet:
 - carbohydrate → 50%
 - fat → 35%
 - protein → 15%
- The requirement is given in 2-4 liter of fluids.
- Types of solutions used
 1. CHO → Glucose 50% + insulin.
 2. Fat → intralipid 10% & 20%.
 3. Protein → vamine or totamine.
- Other elements which has to be added:
 1. K^+ , Mg^{+2} and Cl^-
 2. Trace elements.
 3. Water-soluble vitamins and fat soluble vitamins.
- The fluids are mixed together in a mixer bag & given over 24 hours in CVP.
- Lipids can be given through a peripheral vein because it is isotonic

Important notes about TPN▪ **Intralipid:**

- Fat emulsion produced from soya oil.
- It is isotonic.
- Given in peripheral vein.
- Not to mix it in the same mixture bag.
- Given twice weekly.

▪ **CHO:**

- Raise the dose every 3 – 4 days, regards to ↑ insulin output gradually.

Complications

- Nutritional and metabolic complications resulting in overfeeding, underfeeding or specific nutrient imbalance as hyponatremia, hypokalemia or hyperosmolar dehydration. Hyperglycemia is a common incident.
- **Catheter complications:**
 1. Misplacement of the catheter.
 2. Insertion of a catheter tip in the pleural cavity leading to hemothorax or pneumothorax. Chest x ray should always be done after insertion of a catheter.
 3. Injury to arteries or to nerves of brachial plexus ,rare in experienced hands.
 4. If the infusion set is accidentally detached from the venous catheter air can be sucked into the superior vena cava which have a negative pressure producing serious air embolism.
 5. Venous thrombosis.
 6. A common serious complication is the central venous catheter infection & septic thrombophlebitis which lead to septicemia and if neglected , death, whenever the patient develops unexplained fever that persists for more than 24 hours the venous catheter should be removed and sent for bacteriological and fungal cultures.
- Failure of blood barrier, the absence of enteral feeding leading to atrophy of intestinal mucosa. This allows for translocation of bacteria from the lumen to the blood stream leading to septicemia in the critically ill patients.

Monitoring of the patient

- **Daily:** → urine output, Blood (Urea, Electrolyte, Glucose).
- **Twice weekly:** → Ca, Ph, Albumin, CBC
- **Weekly:** → LFT, Plasma lipids, Magnesium.
- **Monthly:** → foliate, Iron, Zinc, PT

Refeeding Syndrome:

- Severe fluid and electrolyte shift in malnourished patient undergoing refeeding, more common with TPN, but may occur with enteral nutrition.
- Manifested by ↓ Ca, Mg and P → arrhythmia, liver dysfunction, seizures, confusion, ↓ respiratory function, coma up to death.
- Risk patient: alcohol dependant patients, severely malnourished patients.
- Treatment: matching the intake with requirements, avoid overfeeding, regular vitamin administration, Mg and P supplementation.

C. Home Parenteral Nutrition

- Intravenous nutrition at home is usually reserved for patients who had massive small intestinal resection (short bowel \$).
- 1. Permanent silastic central venous catheter, tunneled & cuffed is placed for long-term use.
- 2. Parenteral alimentation, is usually given at night over 12 hours with the aid of mechanical pump.

➡ Metabolic response to starvation:

1. Low plasma insulin.
2. High plasma glucagon.
3. Hepatic glycogenolysis.
4. Protein catabolism.
5. Hepatic gluconeogenesis.
6. Lipolysis: mobilization of fat stores.
7. Adaptive ketogenesis.
8. Reduction in resting energy expenditure.

➡ Metabolic response to trauma & sepsis:

1. ↑ Counter regulatory hormones:
(adrenaline, nor-adrenaline, cortisol, glucagon, growth hormone).
2. ↑ Energy requirements.
3. ↑ Nitrogen requirements.
4. ↑ Gluconeogenesis & protein catabolism
5. Insulin resistance & glucose intolerance.
6. Fluid retention & hypoalbuminemia.

Obesity in Surgery

Definition

- Basal metabolic index (BMI) of more than 30.

Data obtained during recent years shows that the distribution of fat is of greater importance than is the magnitude of obesity. People whose deposits of fat are in the trunk or abdomen (android obesity) have greater morbidity and mortality rates than those with a more typically female distribution on the hips and buttocks

Surgery

Problems of surgery in obese

- **Increased risk of:**
 - Difficult intubation & Aspiration.
 - Myocardial infarction, cerebrovascular accident and DVT.
 - poor wound healing and infection.
 - Bed sores.
 - Mechanical problems (lifting, transferring, operating table & weight limits).

Indications of surgical treatment for obesity

- 1) severely obese (BMI of 40 or more)
- 2) have a BMI of 35 to 39.9 with serious medical conditions (such as high blood cholesterol and triglycerides, hypertension, sleep apnea)
- 3) Tried other methods of weight loss (changes in eating, behavior, increased physical activity and/or drug therapy) and are still severely obese.
- 4) unable to physically perform routine daily activities (work-related and family functions)
- 5) Understand the procedure, risks of surgery and effects after surgery.

Operative procedures

Intestinal bypass and, to a certain extent, gastric bypass Operations limiting the amount of solid food that can be ingested or passed through the upper intestinal tract.

Types of operations

1. Small bowel resection:

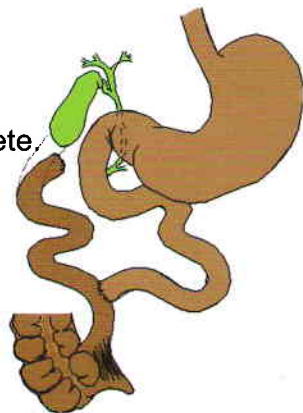
Idea

- weight loss in patients with extensive resections
- The debility and hepatic dysfunction made this procedure obsolete.

2. Jejunioileal bypass

Idea

- A short-bowel syndrome can be created by joining the end of about 35 cm of proximal jejunum to the side of ileum about 10 cm proximal to the ileocolic junction.



End-side jejunioileostomy,

Complications:

- a. Hepatic dysfunction
- b. Hypokalemia .
- c. Hypocalcemia.
- d. Hypomagnesemia.
- e. Renal lithiasis from fat malabsorption.
- f. Oxaluria and dehydration.
- g. Cholelithiasis.
- h. A peculiar form of enteroarthritis.
- i. Prevalent diarrhea.

They are considered to be too high a price to pay.

3. **Biliopancreatic bypass (not widely accepted).**4. **Gastric bypass:****Idea**

- A small gastric pouch is created by stapling across the stomach,
- The gastric pouch is drained by a Roux-en-Y gastrojejunostomy.

Complications:

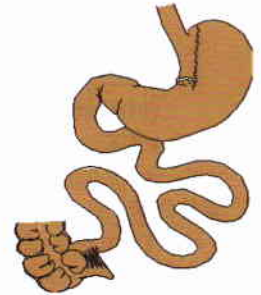
- Deficiencies of iron, calcium, thiamine, vitamin B₁ & B₂, and of folic acid
- Marginal (stomal) ulcers are rare complications.

5. **Vertical banded gastroplasty:**

- Aims to create both a small gastric pouch and an outlet that does not dilate.
- These goals are accomplished by placing four rows of staples parallel to the lesser curvature of the stomach, creating a gastric tube with a capacity of 15 ml or less.

Complications

- 1- Perforations of the esophagus and stomach
- 2- Mortality rates are generally below 1%.



Vertical gastroplasty

Results of antiobesity surgery

1. Around 30 % of preoperative body weight is lost during the first 1 to 2 years.
2. The walls of the stomach are smooth muscle, they expand with time.
3. Compensatory hypertrophy and hyperplasia of the intestine increase absorptive capacity.
4. Obese patients are at increased risk of postoperative complications such as wound infections, hernias, pneumonia, and thromboembolism.

Surgical Hypertension

➡ See in aid to post-graduate book.

Pheochromocytoma

➡ See in endocrine part

Cushing's Syndrome

➡ See in endocrine part

Renovascular Hypertension

➡ See in aid to post-graduate book.

Transplantation

Transplantation Terminology

<u>Autograft:</u>	Donor & recipient is the same person (no rejection).
<u>Isograft:</u>	Donor & recipient are identical twins (no rejection).
<u>Allograft:</u>	Donor & recipient are of same species but genetically different (human to human).
<u>Xenograft :</u>	Donor & recipient are of different species (animal to human).
<u>Orthotopic graft:</u>	A graft placed in its normal anatomical site
<u>Heterotopic graft:</u>	A graft placed in a site different from its normal anatomical site.
<u>Alloantigen:</u>	Transplant antigen
<u>Alloantibody:</u>	Transplant antibody

Immunity in transplantation

⇒ Major Histocompatibility Antigens:

- Glycoprotein on the surface of all somatic cells acts as "self markers" which are responsible for triggering the immune reaction leading to allograft rejection.
- These molecules were originally detected on leucocytes and therefore, named (Human Leukocyte Antigen, HLA).
- HLA are genetically controlled by loci on the short arm of chromosome 6, this area is termed the **major histocompatibility complex**.

⇒ ABO Antigens:

- These antigens are not only present on RBCs but also on most of other cell types.
- There is **no need** to consider **Rh** compatibility in transplantation.

Immune Compatibility Testing:

1. ABO blood grouping and cross matching.
2. HLA cross matching (**important** in kidney, bone marrow and pancreas transplantation while **not important** in heart and liver transplantation).

⇒ Mechanism of graft rejection:

1. **Cellular mechanism:** graft alloantigens → engulfed and processed by macrophages → stimulate T-helper lymphocytes → release interleukin-2 (IL-2) → stimulate cytotoxic T-lymphocytes → attack the transplanted tissues.
2. **Humoral mechanism:** cytotoxic antibodies (present before transplantation e. g. anti-A & anti-B) → complement fixation → attack vascular endothelium in transplanted organ → thrombosis (hyper-acute graft rejection).

⇒ Types of graft rejection:

Type	Onset	Mediator	Treatment	Result
Hyperacute	Immediate	Humoral ABO	Graft removal	Graft loss
Acute	First 6 months	Cellular	High dose steroid OKT3	Good
Chronic(commonest)	After 6 months	Cellular and/or humoral	None (irreversible) Or re-transplantation	Bad

Immune-suppressive therapy

- Immunosuppressive therapy is given to all transplant recipients in order to inhibit the rejection process.
- **Classification according to mode of action:**
 1. **Drugs which deplete circulating lymphocytes:** Corticosteroids, anti-lymphocytic globulin (ALG), Anti-thymocyte globulin (ATG) and monoclonal antibodies (OKT3).
 2. **Drugs which inhibit lymphocyte activation and/or proliferation:** Cyclosporine, tacrolimus (FK-506) and azathioprine.

The three major drugs are corticosteroids (prednisolone), azathioprine (imuran), and cyclosporine.

- **Complications:**
 - A. Complications common for all drugs:
 1. **Infection:** Bacterial, viral (the commonest is CMV), protozoal (the commonest is pneumocystis carinii) and fungal (candida).
 2. **Malignancy:** the commonest is squamous cell carcinoma of skin.
 - B. Specific complications for each drug: e. g.
 1. **Steroids**→ DM, hypertension, weight gain, osteoporosis and peptic ulceration.
 2. **Azathioprine**→ hepatotoxicity and leucopenia.
 3. **Cyclosporine & Tacrolimus**→ nephrotoxicity.

Preparation for transplantation

Types of Organ donors

- **Cadaveric donors (brainstem-dead donors):**
 - Brainstem death is caused by intracranial hemorrhage, trauma, viral/bacterial infection, or a primary intracerebral tumor.
 - The diagnosis is confirmed by CT scan and a series of clinical tests performed twice by two senior doctors establish brainstem.
 - The cadaveric donor should be monitored and supported to maintain the perfusion of tissues to be transplanted (ventilation, plasma and vasopressors).
- **Criteria of brainstem death:** in addition to the flat EEG the following should be checked:
 - The patient must be in apneic coma: The patient is unresponsive & dependent on mechanical ventilation.
 - No corneal reflex
 - No pupillary response to light, direct or consensual
 - Absent gag reflex
 - Absent cough reflex via bronchial stimulation by a catheter in the endotracheal tube.
 - Absent vestibulo – ocular reflex no eye movement upon injection of 50ml of ice cold water into each external auditory meatus.
 - All the previous tests should be repeated after 24 hours and should be done by a separate physician not involved in the transplant procedure
- **Exclusions**
 - **Drug effects:** as sedatives, hypnotics or muscle relaxants
 - **Hypothermia:** core temp must be above 35 °C. Low body temperature can induce deep coma.
 - **Metabolic abnormalities** e.g. hypo or hyperglycemia, hypo or hypernatremia, uremia or hepatic encephalopathy.
 - **Intoxication** by alcohol or other drugs
- **Living donors:**
 - As a healthy first-degree relative (parent, child, or sibling).
 - The donor should be examined, investigated, tissue typed and cross-matched with the recipient.

Organ recipient

- **Investigation of patients for suitability for organ transplantation:**
 - CBC and coagulation profile and blood group.
 - KFTs and LFTs.
 - Glucose and lipid profiles.
 - Chest X-ray and ECG.
 - Viral markers for hepatitis B & C, HIV, CMV, HSV and EBV.
 - **Tissue typing and antibody screening for HLA.**
- **Contraindications of organ transplantation:**
 - Multi-organ failure.
 - Ongoing sepsis.
 - Current malignancy.
 - Active peptic ulceration.

Specific organ transplantation

		<u>Kidney Transplantation</u>	<u>Liver Transplantation</u>	<u>Pancreas Transplantation</u>	<u>Bone Marrow Transplantation</u>
Indications		- <u>Commonest</u> is chronic glomerulonephritis - Chronic pyelonephritis - Lupus nephritis - Hypertension and DM - Polycystic kidneys - Advanced obstructive uropathy	- Cirrhosis (alcoholic, post-viral, biliary or Wilson's) - Biliary atresia (child) - Chronic viral hepatitis - Sclerosing cholangitis - Budd Chiari syndrome - Advanced malignancy	- Type I IDDM + renal failure	- Aplastic anemia - Some types of leukemia
Technique	Graft	Placed extra-peritoneal in the iliac fossa especially the Right Fossa	Placed in the same position (orthotopic transplantation)	Whole or segmental N.B. islets transplantation is still under trials	Bone marrow is aspirated from donor → filtration & heparinization Then, IV infusion to the recipient
	Anastomosis	• <u>Artery</u>→ renal with external iliac (end-to-side) • <u>Vein</u>→ renal with external iliac (end-to-side) • <u>Ureter</u>→ to urinary bladder under mucosal tunnel	• Vessels and ducts are anastomosed to their corresponding	• <u>Veins</u>→ to either portal or systemic circulation • <u>Ducts</u>→ to the gall bladder or GIT (by Roux-en-Y)	
Complications		1. Anastomotic leakage or stricture 2. Rejection 3. Recurrence of original disease 4. Complications of immune-suppression (mention)			
Prognosis		1 YSR→ 95% if cadaveric → 98% if living donor 5 YSR→ 60% if cadaveric → 90% if living donor	<u>1 YSR</u> → 85% <u>5 YSR</u> → 50%	<u>1 YSR</u> → 45%	

Heart transplantation

- ➡ **Indications:** 1) Advanced CAD not suitable for coronary bypass. 2) Idiopathic cardiomyopathy
 (Combined Ht. & lung transplantation may be done if both organs are damaged)
- ➡ **Complications:** 1) Of immunosuppressive. 2) Graft rejection 3) Graft atherosclerosis

Imaging in Surgery

Ultrasound (SONAR) = Sound on navigation and Ranging

Definition

- Sound waves at frequency between 2.5 - 7.5 MHz, which is more than what the human ear can hear.

Uses

- **Diagnostic:**
 - Easy, non-invasive and can be used Intra-operative (e.g. laparoscopic U/S).
 - Guided biopsy.
- **Therapeutic:**
 - Guided drainage.
 - Renal stones.

Disadvantages

- **Poor in:**
 - (Gas) e.g. colon → solved by laparoscopic US and endoscopic US
 - Fat people

Application

- 1- **Gall bladder:** calculi, shape, size, acute and chronic cholecystitis.
- 2- **Liver and spleen:** rupture, shape, size, acute and chronic hepatitis, abscess.
- 3- **Shunt surgery:** preoperative → localization of renal vein and pattern of splenic vein.
- 4- **Jaundice:** 1st radiological investigation → intra and extra hepatic biliary system.
- 5- **Ascites:** free fluid in hepato-renal pouch or in flanks.
- 6- **Acute abdomen:** help in detection of causes e.g. ruptured cyst.
- 7- **Colon:** bad in diagnosis but may detect a mass.
- 8- **Stomach:** bad in diagnosis but may detect mass if > 3 cm.
- 9- **Anterior abdominal wall** → desmoid tumor.
- 10- **Retro-peritoneal tumors.**
- 11- **Kidney:**
 - Calculi, site, size, dilatation (calyces, pelvis and hydro and pyonephrosis).
 - Cyst (solitary, polycystic kidneys).
 - Ectopic kidney.
 - Peri-nephric abscess.
 - Chronic glomerulonephritis.
- 12- **Ureters:** dilatation or stones.
- 13- **Urinary bladder:** Stone, mass, diverticulae or cystitis.
- 14- **Prostate:** size, pattern, BPH, cancer (better by trans-rectal US).
- 15- **Testis:** tumors, undescended testis, spermatocele, hydro, pyo or hematocele.
- 16- **Vascular:**
 - Aorta: AAA if > 3 cm.
 - Hepatic vein, portal vein and splenic vein.
 - Renal blood vessels.
- 17- **Breast:**
 - Mass (if solid and > 2 cm → echogenic).
 - Cysts (e.g. abscess → sonolucent).
- 18- **Thyroid:** solid mass, cyst.
- 19- **Salivary glands:** stone, mass.
- 20- **Chest:** diaphragmatic movement, subphrenic collection, pleural effusion and mass.

Laparoscopy in Surgery

Advantages

- Less pain.
- Less hospital stay.
- Early ambulence.
- Better cosmetically.

Indications

1- Diagnostic:

A- Elective:

- Liver, GB diseases (trans-hepatic, trans-cystic).
- Spleen: hepato-splenomegaly.
- Oncology: staging and biopsy.

B- Emergency:

- Abdominal trauma, acute abdomen, adhesions.

2- Therapeutic:

- Laparoscopic cholecystectomy, appendectomy, varicocelelectomy, vagotomy, nephrectomy and cystectomy (UB).
- Adhesiolysis.
- Herniorrhaphy or hernioplasty.

Contraindications

A- major:

- Pregnancy, paralytic ileus and generalized peritonitis.
- Coagulation disorders, cardiac and respiratory disease.

B- Minor:

- Obesity.
- Hiatus hernia.
- Irreducible external hernia.

C- Specific:

- Appendectomy → phlegmon of caecal wall perforation near the base.
- Carcinoid syndrome.

Preoperative preparation

- Inform patient (consent).
- Anesthesia.
- NGT.
- Clean the umbilicus.

Preoperative medication

- Atropine → ↓ vagal tone.
- Diazepam → ↓ anxiety.

Anesthesia

- GA, regional Anesthesia (according to type of operation).

Principle

- a- Pneumo-peritoneum by NO₂ or CO₂ (better) → improves vision & decrease tissue trauma.
- b- 4 skin stabs (½ - 1 cm) → 1 for camera and 3 for instruments.
- c- Inspect the abdomen → then complete the operation.
- d- Remove instruments → deflation → clean wound (iodine or alcohol).
- e- Close by 3/0 or 4/0 silk suture.

Complications

Intra-operative

- Emphysema of skin, omentum, mediastinum and gas embolism.
- Blood vessel injury: epigastric BVs, IVC.
- Hollow viscus lesion: GB, intestine → escape of contents → peritonitis.
- Loss of foreign body e.g. clips.

Postoperative

- Delayed hemorrhage and late discovery of hollow organ injury (UB, colon).
- DVT (↑ risk of DVT in laparoscopic surgery in urology > general).
- Postoperative shoulder pain.

Radio-isotope Scans (Nuclear Medicine)

Diagnosis

- Most commonly used is Tc^{99}
- **Others:**
 - Heart → $Thalium^{201}$ → myocardial infarction.
 - Gallium-citrate (Ga^{67}) → tumor, infection, inflammation.
 - Thyroid → iodine (I^{121}).
 - Blood cells (chromium Cr^{51}) → RBCs sequestration in spleen.

Computed Tomography

Technique

- X-ray tube around the patient emitting radiation beams with serial X-rays → computer will display it as regularly-spaced cross sections.
- Air will appear dark, and bone will appear white.

Advantages

- More accurate, clear and some lesions are better diagnosed by it

Disadvantages

- Expensive and hazardous

Magnetic Resonance Imaging (MRI)

- Nuclear magnetization (by nuclei with odd number of protons e.g. hydrogen, carbon or phosphorus, placed in a magnetic field).

Technique

- Radiofrequency pulses emitting signals to the computer → change it into image.

Advantages

- Gives better resolution for soft tissue than CT, so it is the first investigation for brain tumors, cerebral hemorrhage and infection.
- Also in: neck, mediastinum, abdomen, pelvis and orthopedics.

Disadvantages

- In spite of being not biologically hazardous, it is contraindicated in cardiac pace makers, metal prosthetics.

LASER in Surgery

➡ See in aid to post-graduate book.

Clinical Audit

Definition

Process used by clinicians who seek to improve patient care via comparing aspects of care (structure, process, outcome) against explicit criteria.

Structure

- The quantity & type of available resources.

Process

- Define what is done to the patient:
 - a. Adequate preoperative assessment.
 - b. The way an operation was performed.
 - c. Adequate documentation.
 - d. Compliance with policies of the hospital and/or international practice.

Outcome

- The result of the clinical intervention, either success or failure should be documented
 - a. Morbidity and mortality meetings (a conclusion should be reached as regards the cause & its avoidance).
 - b. Patient satisfaction.

Hints about audit

- Adequate & accurate medical recording is important. It should be in way agreed by the treating physicians and administration.
- It is an organized team approach.
- It is well-known that errors do occur in medical practice, the auditing process aims to minimizing the errors & high quality medical care is the target.
- Comparative audit: to compare the outcome of institute with other centers.
- The auditing process have educational advantages.

Surgical Ethics

Introduction

- Ethics and surgical intervention must go hand in hand.
- **The difference between criminal and surgeon is that:**
 - The surgeon causes harm only incidentally.
 - The surgeon's intervention is to cure or manage illness.
 - Any bodily invasion occurs only with the permission of the patient.
- **The 2 general duties of surgical care are:**
 - Protect life and health of the patients.
 - Respect autonomy of the patients and their ability to make choices for the treatment.
- **The specific duties of surgeons :**
 - Acceptable practice concerning informed consent.
 - Confidentiality.
 - Decisions not to provide or to omit life sustaining care.
 - Surgical research and the maintenance of good professional standards.

Informed Consent

- In surgical practice, respect for autonomy is to obtain informed consent before beginning of the treatment.
- **Information that patients need to know before consent are:**
 - The condition and the reasons why the surgery is needed.
 - The type of surgery and how it may correct the condition.
 - The anticipate prognosis and expected side effects of the proposed surgery.
 - Any alternative and potentially successful treatment other than surgery.
 - The consequences of no treatment at all.
- **For consent to be valid patients must be:**
 - Competent to give the consent, able to understand, remember and be cautious about information provided about treatment choices.
 - Not to be forced into decisions.
 - Given sufficient information about treatment choices.
- **Surgeons will face 3 practical difficulties while achieving these goals:**
 1. It is necessary to obtain informed consent every time a patient is touched during their care.
 - ➔ Obtain proper and clear consent in the 1st place.
 2. Patient's inability to give consent because of temporary unconsciousness while patient may be at risk of death or permanent disability if surgery is not immediately performed.
 - ➔ The situation is one of medical necessity and intervention can occur without consent unless the patient had made a legally valid advance decision refusing such specific treatment.
 3. Incompetent patient (المريض فاقد الأهلية)
 - a. Children:
 - In this case parents are required to give written consent.
 - Take care to explain to children what is surgically proposed.
 - Even some young children can be competent to do consent.
 - b. Psychiatric ill or mentally handicapped patients:
 - Those who are responsible for them will make the consent.

Confidentiality

- Respect for autonomy includes respect of patient privacy. Such respect means that surgeons must not discuss clinical matters with others without patient consent.
- However, surgeons are allowed to use private information with other professionals who are part of the health care team if this directly affects the treatment plan.
- Research and maintaining standards of healthcare is a part of surgeon's duty.
- To maintain an acceptable standard, surgeons must acquire further education and training throughout their career.
- Surgeons also have a duty to monitor the performance of their colleagues.
- If necessary, faulty surgeons must be stopped from practicing until they can undergo further appropriate training and counseling.

Surgical Drains

➡ **See surgical instrument book.**